Interpretive Summary of Existing Data Relevant to Potential Contaminants of Concern within the Anacostia River Watershed

Prepared by:

Syracuse Research Corporation
6225 Running Ridge Road
North Syracuse, NY 13212
June 2000

National Oceanic and Atmospheric Administration
Coastal Protection and Restoration Division
7600 Sandpoint Way N.E.
Seattle, WA 98115
June 2000

Prepared for:

GEO-CENTERS, Inc.
4555 Overlook Avenue, S.W.
Building 207, Room 302
Washington, DC 20375

Attention:
Richard Beers, Project Manager
202-404-6297

Contract # GC-3381-99-002
SRC # FA292

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ACRONYMS FOR ANACOSTIA FILES

ARBC	Anacostia River business coalition
AWCAC	Anacostia Watershed Citizens Advisory Committee
AWRC	Anacostia Watershed Restoration Committee
CBFO	Chesapeake Bay Field Office
COG	Metropolitan Washington Council of Governments
CSO	Combined Sewer Overflow
CWA	Clean Water Act
DNT	Maryland Department of Natural Resources
DCRA	Department of Consumer Regulatory Affairs
GDC	Government of the District of Columbia
ICPRB	Interstate Commission on the Potomac River Basin
MDE	Maryland Department of the Environment
MWCOG	Metropolitan Washington Council of Governments
NCP	National Capital Parks
NPS	National Parks Service
NPDES	National Pollutant Discharge Elimination System
USACE	United States Army Corps of Engineers
USCOE	United States Army Corps of Engineers
U.S. EPA-CBP	United States Environmental Protection Agency and Chesapeake Bay Program
USFWS	U.S. Fish and Wildlife Service

PREFACE

This report was prepared by Syracuse Research Corporation (SRC) under a contract from GEO-CENTERS, Inc (GC-3381-99-002) for the Anacostia Watershed Toxics Alliance. The report was developed in collaboration with the National Oceanic and Atmospheric Administration (NOAA) and O'Brien and Gere Engineers, Inc. Contributions made by NOAA included assembling all of the environmental monitoring data into the Anacostia River Watershed Database and Mapping Project, the development of Chapter 7, which describes the ecological screening-level risk assessment, and related contributions to Chapters 8 and 9. Contributions made by O'Brien and Gere Engineers included the evaluation of information on river hydrodynamics and sediment transport, and development of Chapters 3 and 4. Important contributions were also made by U.S. EPA Region 3, which provided access to many of the background reports and documents that were reviewed, and by the Anacostia Watershed Toxics Alliance, whose members provided comments and suggestions on an earlier draft of the report (March 2000).

1. INTRODUCTION

The Anacostia Watershed Toxics Alliance (AWTA) was created to foster a public-private partnership in establishing a watershed-wide focus on the assessment and management of toxic chemicals in the Anacostia River. The mission of the AWTA is as follows:

> *To work in good faith as partners to evaluate the presence, sources, and impacts of toxic contaminants on the Anacostia River with all stakeholders, both public and private, and other interested parties, and to evaluate and take actions to enhance the restoration of the watershed to its beneficial use to the community and ecosystem as a whole.*

To achieve this mission, the AWTA has adopted the following objectives: 1) Identify and quantitatively assess risks to human health and the environment from toxic contaminants in the Anacostia River. 2) Reduce risks from toxic contaminants to levels that are safe for humans and aquatic life. 3) Build effective partnerships among AWTA members, encourage public input and promote effective restoration of the Anacostia watershed.

As part of activities directed at the above objectives, an assessment of chemical hazards and characterization of related risks to humans and ecological receptors was initiated. This report summarizes the results of Phase I of the risk characterization. The objective of Phase I was to summarize, within a generally accepted data quality evaluation framework, information about the historical and current conditions and concerns related to the environmental quality of the Anacostia Watershed. The currently available information was to form the basis of the following: 1) a preliminary conceptual site model of the tidal Anacostia River and associated chemical hazards; 2) screening-level human health and ecological risk assessments to identify chemicals of potential concern (COPCs) in the tidal Anacostia River; and 3) identification of data gaps relevant to developing a more complete risk characterization of the tidal Anacostia and evaluating potential remediation alternatives, should they be needed. Phase I was to be followed with Phase II data collection efforts to fill data gaps identified in Phase I.

The data quality evaluation framework used in this project is that described in the Risk Assessment Guidelines for Superfund (RAGS), Volume 1, Part A, Chapter 5: Data Evaluation (U.S. EPA, 1989; 1997), as modified by the U.S. EPA Region III Technical Guidance Manual, Selecting Exposure Routes and Contaminants of Concern by Risk-based Screening (U.S. EPA, 1993a), and the Interim Final Guidance on Data Quality Objectives Process for Superfund (U.S. EPA, 1993b). Components of the process used in the screening assessment, and how it would link to Phase II, or subsequent remedial investigation and baseline ecological and human health risk assessments are depicted in Figure 1-1.

The Phase I assessment began with collecting and organizing the existing information about watershed usage, sources of contamination, processes for contaminant deposition and transport, potential exposure pathways and receptors. The information used in the assessment was that made available by the AWTA and other sources prior to January, 2000. This included data in the Anacostia River Watershed Database and Mapping Project developed by the National Oceanic and Atmospheric Administration (NOAA, 2000) and water quality data that was provided by NOAA in spreadsheet format on January 3, 2000; the latter is from a study by Velinsky et al. (1999). Subsequent to completing a preliminary draft of the Phase I report in March, 2000, additional data, information and reports were identified by the ATWTA that would have been of value for consideration in Phase I. However, in order to accommodate the logistical

demands of completing Phase 1 in a timely manner, this additional information could not be included in all aspects of the Phase I assessment. This information will be considered in evaluating the data gaps identified in Phase I and in subsequent reassessments that may occur as part of Phase II activities.

This report is organized into ten chapters and three appendices. Chapter 2 summarizes information on the natural history and environmental management of the Anacostia Watershed. Chapters 3 and 4 summarize information on hydrodynamics and sediment transport in the tidal Anacostia. Chapter 5 describes the data that was used in screening level assessments. The results of the human health and ecological screening assessments are provided in Chapters 6 and 7, respectively. Chapters 8 presents the major conclusions of the assessment, data gaps relevant to fully characterizing human health and ecological risks, including those related to completing a conceptual site model and the development of hydrodynamic and sediment transport models. Recommendations for future data collection and assessment activities are presented in Chapter 9. Supporting data tables, hazard identification summaries for COPCs, and RAGS Section D tables are provided in the appendices.

REFERENCES FOR CHAPTER 1

NOAA. 2000. Anacostia River Watershed Database and Mapping Project. Release 1. National Oceanic and Atmospheric Administration, Office of Response and Restoration, Coastal Protection and Restoration Division.

U.S. EPA. 1989. Risk Assessment Guidelines for Superfund Volume 1, Human Health Evaluation Manual (Part A). U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA/540/1-89/002.

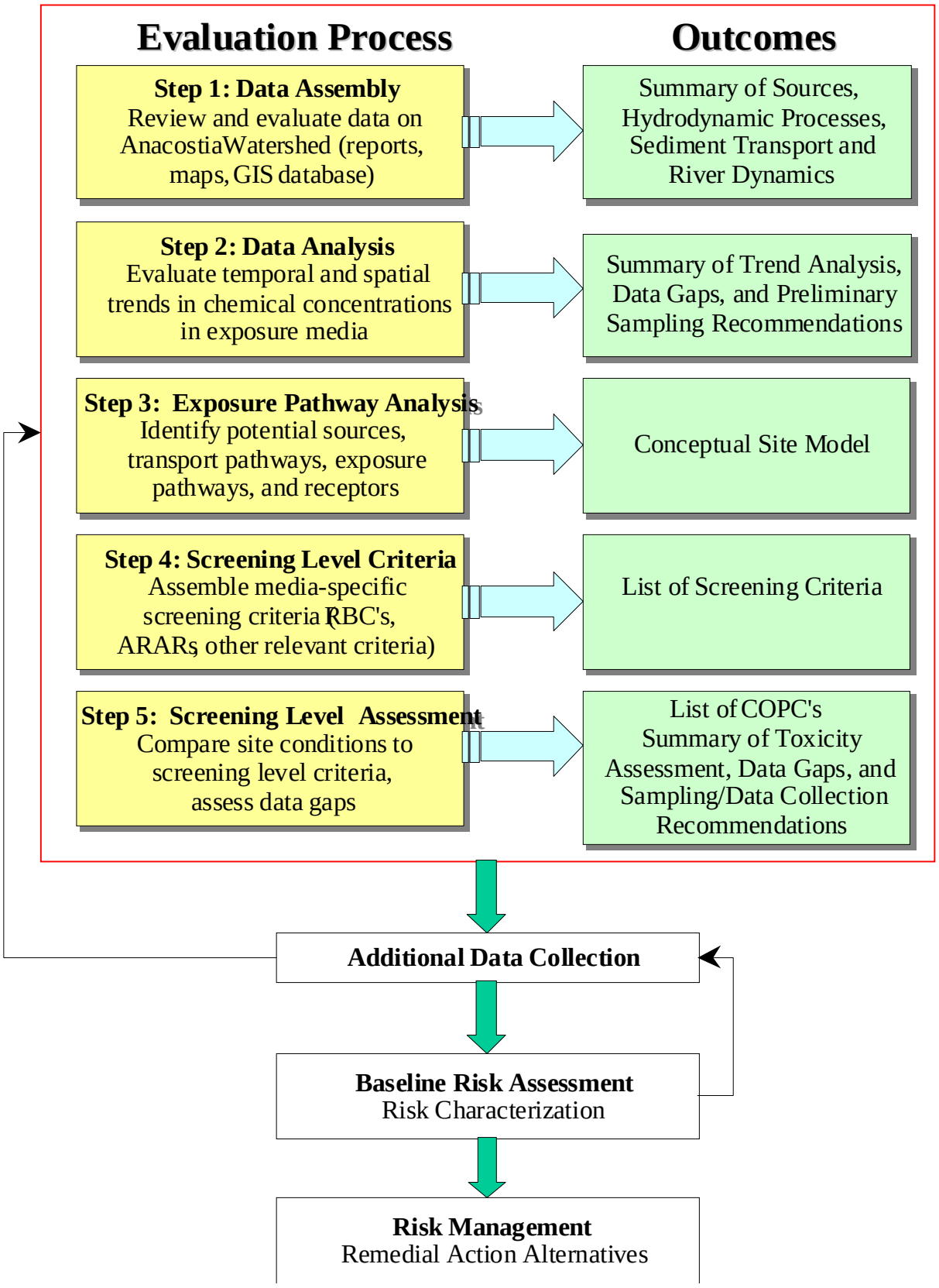
U.S. EPA. 1993a. EPA Region 3 Technical Guidance Manual, Selecting Exposure Routes and Contaminants of Concern by Risk-based Screening. U.S. Environmental Protection Agency, Region III. EPA/903/R-93-001.

U.S. EPA. 1993b. Data Quality Objectives Process for Superfund. Interim Final Guidance. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. EPA540-R-93-071.

U.S. EPA. 1997. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments, interim final. EPA 540-R-97-006. U.S. Environmental Protection Agency, Environmental Response Team, Edison, NJ.

Velinsky, D.J., G.F. Riedel, and G. Foster. 1999. Effects of stormwater runoff on the water quality of the tidal Anacostia River. Final Report 8PO-331-NAEX ed. U.S. EPA, Region III, Philadelphia. 132 pages.

Figure 1-1. Overview of Process and Outcomes of Screening Level Assessment [REED].



2. BACKGROUND ON THE ANACOSTIA WATERSHED

2.1 NATURAL HISTORY AND HISTORICAL CHANGES IN HUMAN USES OF THE ANACOSTIA RIVER WATERSHED

2.1.1 PHYSIOGRAPHY

The Anacostia River watershed drains approximately 403 km^2 of land area (Scatena, 1986). It is comprised of the Piedmont Plateau and Atlantic Coastal Plain physiographic provinces, divided at the Fall Line, which is an area of steep descent in the watershed streams from the plateau to the plain (U.S. EPA-CBP, 1992). The Northwest Branch of the Anacostia River drains a 127 km^2 area of ridge and ravine topography carved into the Piedmont crystalline metamorphic rock of the plateau, and the Northeast Branch drains a 190 km^2 area of primarily relatively flat coastal plain sediments comprised of alternating layers of sand, silt, and clay (Scatena, 1986). The channel of the tidal Anacostia River occupies a narrow strip between Pleistocene terraces (Scatena, 1986).

2.1.2 CLIMATE AND HYDROLOGY

The Anacostia River watershed is a sub-watershed within the Potomac River Drainage Basin, which in turn empties into the Chesapeake Bay (U.S. EPA-CBP, 1992). The climate of the region is characterized by hot summer and mild winter temperatures, and precipitation is evenly distributed throughout the year, averaging approximately 106 cm/year in the center of the watershed (Scatena, 1986). Most of the rainfall in the summer is provided by intense, convective thunderstorm events (Metropolitan Washington Council of Governments [MWCOG], 1986).

The mean yearly discharge of the Anacostia River is 3.9 m^3/sec, and records as of 1986 indicated a minimum discharge of 0.05 m^3/sec and a maximum of 900 m^3/sec (Scatena, 1986). The high volume to influx ratio in the tidal Anacostia River results in a flow rate that is frequently described as *sluggish*. One estimate of the residence time of water in the tidal Anacostia River was 35 days (Scatena, 1986), and another report estimated residence times of between 20 and 40 days under average rainfall conditions, and 100 days or more during periods of low rainfall (MWCOG, 1986).

The waters of the Anacostia River originate in northern Prince Georges and Montgomery counties in Maryland, and flow freely through numerous tributaries to the confluence of the Northeast and Northwest Branches, where the freshwater, tidal Anacostia River is formed (U.S. EPA, 1999a). Several other smaller tributaries empty directly into the tidal Anacostia along its length (U.S. EPA, 1999a). Sand bars and shoals near the mouths of tidal Anacostia tributaries provide evidence of upstream erosion (MWCOG, 1986).

The current channel of the tidal Anacostia River, as well as the Kingman Lake and Kenilworth Marsh embayments, are almost entirely man-made (Scatena, 1986). Efforts during the period 1902 to 1960 to implement flood control projects, construct sea walls on both sides of the tidal Anacostia River, and fill in wetlands ultimately contributed to ecological degradation by eliminating extensive areas of wetland, aquatic habitat, and bottomland hardwood forest (U.S. EPA, 1999a). Natural wetlands and riverside forests can act as an effective ecological buffer, erosion control, and filtration systems between populated areas and a major water body.

The tidal Anacostia River is both free-flowing and tidally influenced. During storms, the waterway behaves like a river with unidirectional, down-gradient flow, whereas, between storm events, the Anacostia behaves more like a tidal lake with 3-foot tidal fluctuations (Scatena, 1986). Coffin et al. (1998, 1999) attributed thermal stratification observed during June 1997 through February 1998 in the center of the lower Anacostia River (between the Washington Naval Yard and the Pennsylvania Avenue Bridge) to tidal circulation, which may periodically move cooler Potomac River water into the lower surface water stratum of the lower Anacostia River.

2.1.3 LAND USE/HUMAN USE

At the time of European colonization of the Atlantic coast during the early 1600s, the area surrounding the confluence of the Potomac and Anacostia Rivers was forested and used by the native Nacotchtank Indians for subsistence hunting and as a regional trading center (U.S. EPA, 1999a). The Anacostia waterway itself reportedly contained abundant populations of numerous species of fish, including the sturgeon, American and hickory chad, white and yellow perch, red-breasted sunfish, striped bass, catfish, and herring and was utilized extensively for subsistence fishing by the native people and early settlers (U.S. EPA, 1999a).

As the area was more heavily settled by European immigrants, the Anacostia River became an early shipping and trade region and was navigable throughout the length of the 8.4-mile tidal portion of the river up to the historic port city of Bladensburg, MD, near the confluence of the Northeast and Northwest Branches (U.S. EPA, 1999a, Warner et al., 1997).

Widespread deforestation for tobacco farming in colonial times and later commercial crop farming during the late 1700s and early 1800s contributed to massive surface soil erosion and heavy inputs of sediments to the tidal river, so that by the mid 1800s, ships could no longer navigate the upper tidal river to the port at Bladensburg (Kumble, 1990 ; U.S. EPA, 1999a), and extensive mud flats formed along the shoreline (Kumble, 1990). More recently, surface mining excavations and urban/suburban development continued to contribute to deforestation, resulting in estimated sediment loads to the tidal Anacostia River of 134,420 tons/year in 1963 and 137,000 tons/year in 1981 (Scatena, 1986). Surface mining operations and abandoned sand and gravel mines comprise approximately only 2% of the land area in the Maryland portion of the Anacostia watershed (U.S. EPA-CBP, 1992), but they are suspected of contributing significantly to the current total annual loading of sediment to the tidal Anacostia River (MWCOG, 1986 ; Warner et al., 1997).

Based on an analysis of aerial photographs taken in 1990, only 7.1% of the entire watershed remained in agriculture, 1.6% was sand and gravel mines, 43.3% was residential, and a significant fraction of the watershed was in forest (24.6%), parkland (7.1%), or wetland (2.8%); the remaining 16.7% of land area was accounted for as *institutional*, *commercial*, *industrial*, or *federal* use categories (Warner et al., 1997). Most of the agricultural area lies within the USDA Beltsville Agricultural Research Center within the Northeast Branch drainage area, and most of the forest area is confined to the headwaters of the tributary streams (Scatena, 1986).

2.1.4 URBANIZATION

The land area comprising the Anacostia River watershed falls within the local jurisdictions of Prince Georges and Montgomery Counties of the state of Maryland and the District of Columbia, and most of the tidal Anacostia River falls within the boundaries of the District of Columbia (U.S. EPA-CBP, 1992).

Over the period 1980 to 1990, the populations of Prince Georges and Montgomery Counties increased

9.7 and 30.7%, respectively, and the number of housing units has increased by 14.2 and 36.8%, respectively (U.S. EPA-CBP, 1992). More than 800,000 people currently live in the Anacostia River watershed, with the highest density of residents near the tidal Anacostia (U.S. EPA, 1999a). As in other highly populated urban river areas, continuous potential sources of pollution include human and industrial waste, trash, and petroleum-based chemical runoff from intensive vehicular traffic. These challenges are particularly difficult in some urban areas along the tidal Anacostia River. For example, antiquated combined sewer systems designed for human populations and land use conditions of nearly a century ago are overtaxed and frequently overflow during storms, emptying untreated sewage directly into the river (U.S. EPA-CBP, 1992).

The land area within the tidal portion of the Anacostia River watershed is almost entirely developed, with scattered woodlands in parks (U.S. EPA-CBP, 1992). Suburban residential settlement and commercial development has also continued in the upper watershed, contributing to deforestation and streambank erosion. Development throughout the watershed has resulted in a high proportion of the land area in impervious surfaces (e.g., pavement in roads and parking lots, sidewalks, and residential/ commercial/ industrial structures), which adversely impacts stream hydrology, water quality, and ecology (U.S. EPA-CBP, 1992). Much of the impervious surface is drained by collecting water from large surface areas (water that otherwise would have soaked into the ground, recharging the groundwater) into narrow outflow channels, which increases downstream flow velocity and leads to erosion, especially during storm events. Hydrographs obtained from USGS gauging stations in 1988–1989 indicate that flow is extremely responsive to even small rainfall events, which appears to be a direct result of increased impervious surfaces from urbanization (Kumble, 1990).

2.2 AN OVERVIEW OF THE ENVIRONMENTAL ISSUES IN THE ANACOSTIA RIVER WATERSHED AND PROGRAMS DEVELOPED TO STUDY OR MANAGE THE WATERSHED

2.2.1 RATINGS OF OVERALL ENVIRONMENTAL QUALITY IN THE ANACOSTIA RIVER

The environmental quality of the Anacostia River has consistently been rated poorly relative to other water bodies in evaluations by several different organizations. The unanimously poor ratings of the Anacostia River and most of its watershed have been based on both ecological and human health issues.

The District of Columbia prepared a draft unified watershed assessment report in 1998 concerning the waters of the District, which included part of the Anacostia watershed (MWCOG, 1998). The assessment included a Nonpoint Source Management Program Watershed Priority List, which rated the Anacostia River and some of its tributaries, specifically Watts Branch, Hickey Run, and Kingman Lake, as *high priority* water bodies (using a ranking system of high, medium, and low priority). These high priority water bodies were considered to be most in need of restoration and were also considered for review for possible accelerated restoration.

The 1998 Maryland Clean Water Action Plan Technical Workgroup's *Report on Unified Watershed Assessment, Watershed Prioritization and Plans for Restoration Action Strategies* identified Category 1 priority watersheds in Maryland that do not meet general water quality or other natural resource goals, and which have been recommended for restoration action within the next two years. The Anacostia River watershed was identified as a priority restoration watershed based on a low non-tidal benthic *Index of Biotic Integrity*, high percent impervious surface, high population density, and high soil erodability (Clean Water Action Plan Technical Work Group, 1998).

The U.S. EPA has identified the Anacostia River as one of the most contaminated rivers in the Chesapeake Bay watershed (AWRC, 1999); it is one of only three sites recognized by the U.S. EPA's Chesapeake Bay Program as posing a significant risk to aquatic life due to sediment contamination, and

has been designated a *Region of Concern* based, in part, on the issuance of a fish consumption advisory for elevated levels of PCBs and chlordane in fish (Government of the District of Columbia [GDC], 1996 ; MWCOG, 1998).

The Anacostia River is widely regarded to be among the 10 most polluted urban rivers in the United States (GDC, 1994, 1996, 1998), and the American Rivers conservation organization identified the Anacostia River as the fourth most polluted river in the United States as of 1993 (MWCOG, 1998) and the seventh most polluted river in 1997. During 1993–1994, a White House Ecosystem Management Task Force completed a case study of the Anacostia River, one of only seven in the nation, and recommended a substantial and coordinated federal role in restoring the watershed (U.S. EPA, 1999).

2.2.2 MAJOR ENVIRONMENTAL QUALITY ISSUES IN THE ANACOSTIA RIVER WATERSHED

Numerous reports have identified major environmental pollution issues in the Anacostia River watershed (e.g., Kumble, 1990 ; McCabe, 1997 ; MWCOG, 1986, 1991, 1998 ; U.S. EPA-CBP, 1992). A brief review of the nature of physical, biological, and chemical stressors to the environmental integrity of the system based on these and other reports, is provided here. Other important environmental issues have also been identified concerning the Anacostia watershed, including drinking water, land use, public education, community involvement, resource use, funding for environmental programs, and environmental justice; these are not addressed in this review.

2.2.2.1 PHYSICAL AND BIOLOGICAL STRESSORS

Major physical stressors include a persistently high particulate loading to the tidal Anacostia River, particularly during storm events, leading to high turbidity and high rates of sedimentation, both of which seriously impact the viability of submerged biotic communities and potentially contribute to the transport of biological and chemical agents within the watershed. The suspended particulates are reported to originate from storm-related erosion and non-point surface runoff in the upper tributaries and from non-point source runoff (both direct inflow and via stormwater catchments) from impervious surfaces in the tidal Anacostia. The absence of adequately vegetated upland and wetland buffer areas in some areas of the watershed also contributes to the problem of particulate loadings from non-point surface runoff.

Biological stressors in the Anacostia River include fecal coliform (and possibly other pathogens) originating from the repeated and significant influx of untreated sewage to the tidal Anacostia River from outdated combined sewer overflows (CSOs) during storm events. Due in part to the persistent presence of significant levels of fecal coliform bacteria, the tidal Anacostia River is not currently classified as a Class A (primary contact) waterbody under the Clean Water Act system of classification, although it is slated for future Class A designation, presumably pending successful restoration (GDC, 1994, 1996, 1998). High nutrient loadings to the Anacostia from non-point surface runoff and CSOs also potentially contribute to biological stress by promoting eutrophication, which may lead to dissolved oxygen depletion sufficient to impact water column and benthic biota.

2.2.2.2 CHEMICAL STRESSORS

Numerous reports concur that major pollution issues concerning the Anacostia River watershed include non-point source loadings, combined sewer overflows, high rates of erosion and downstream sedimentation, high nutrient loadings, and chemical contamination of sediments and fish. While each type of stressor may contribute significantly to overall environmental degradation, this screening level risk assessment is concerned primarily with the potential for adverse health effects in human and ecological receptors from exposures to chemicals in the Anacostia River.

Many studies have identified the presence of chemical contaminants in sediment, surface water, or fish (e.g., Banta and Horowitz, 1992 ; Banta, 1993 ; Block, 1990 Clark and Crutchley, 1995 ; Clark and Gower, 1995 ; Coffin et al., 1998 ; Coffin et al., 1999 ; GDC, 1998 ; Groundwater and Environmental Services, 1998 ; Gruessner et al., 1997 ; Harshberger et al., 1997 ; Haywood and Focazio, 1990 ; Herson-Jones et al., 1994 ; Hydro-Terra, Inc., 1989, 1997 ; Loos, 1999 ; LTI, 1990 ; McCabe, 1997 ; MWCOG, 1986 ; Montaser, 1997 ; Phelps, 1985, no date ; Phelps and Clark, 1988 ; Pinkney et al., 1993 ; Pinkney, 1999 ; Shepp and Cole, 1993 ; Shepp and Parikh, 1995 ; Stribling et al., 1999 ; U.S. EPA-CBP, 1992 ; USFWS, 1990, 1994 ; Velinsky et al., 1992, 1994, 1999 ; Velinsky and Cummins, 1994, 1996 ; Wade et al., 1994).

Due to PCB and chlordane concentrations in fish above the FDA action levels of 2.0 and 0.3 ppm, respectively, a fish consumption advisory was first issued in 1989 for the District of Columbia's portion of the Anacostia River (GDC, 1994). The advisory was re-issued in 1994 to discourage consumption of any bottom dwelling fish, specifically catfish, eel, and carp, to recommend strict limits on weekly adult consumption rates of predatory game fish such as largemouth bass, and to identify pregnant women and children as groups particularly at risk from consuming Anacostia River fish (MWCOG, 1998). Using a risk assessment approach, ATSDR (1998) concluded that concentrations of contaminants, particularly PCBs, in fish collected for analysis in 1991 from the Anacostia and Potomac Rivers could pose a public health hazard for sport fishermen.

In spite of the fish advisories, consumption of contaminated fish is potentially a significant route of human exposure to chemicals in the tidal Anacostia river. As an indication of fishing use of District of Columbia waters, including the Anacostia River, the number of fishing licenses sold in the District more than doubled during the years 1988–1997 (GDC, 1998). Two surveys of anglers conducted in the early 1990s indicated that fish caught in the tidal Anacostia River, including species specified in fish advisories, were regularly consumed as a source of food by a substantial proportion of shoreline anglers (McCabe, 1997).

The control of hazardous chemical loadings has been regarded as one of the primary issues of concern in the Anacostia River in recent comprehensive reviews of pollution issues in the Anacostia watershed (ICPRB, 1996 ; McCabe, 1997).

A comprehensive study of chemical contaminant sources throughout the Anacostia watershed prepared by the District of Columbia identified nonpoint sources as the primary concern, although the study acknowledged that no information concerning groundwater or direct aerial deposition contributions, illicit dumping or discharge, or CERCLA/Superfund sites was used in the evaluation (Warner et al., 1997). With respect to chemical contaminants in the Anacostia watershed, the source report addressed loadings of petroleum hydrocarbons, lead, and zinc, in particular. Hickey Run was reported to have a history of episodic petroleum hydrocarbon inputs. A plan to trace the specific point sources in Hickey Run is being implemented by MWCOG (Warner et al., 1997). Eighty-five percent of the annual loadings of lead were from CSOs in the tidal Anacostia, while zinc loadings were predominantly from nonpoint sources (Warner et al., 1997). The loadings of arsenic, chromium, and copper and chlordane were also attributed to nonpoint sources, although the study reported that no data concerning CSO loadings were available for these metals. *No other specific chemicals were evaluated in the Warner et al. (1997) source study*. The Interstate Commission on the Potomac River Basin (ICPRB) identified surface runoff, storm and combined sewer discharges, and direct atmospheric deposition as potential sources of hazardous substances detected in sediment sampled in the tidal Anacostia River (Gruessner et al., 1997). Particulates that were aerially deposited on impervious surfaces throughout the watershed are likely to contribute substantially to total chemical loading to the tidal river surface waters via stormwater runoff (ICPRB, 1996; Warner et al., 1997). The relative contribution of direct aerial deposition to total loadings of chemicals to the surface waters of the tidal Anacostia is uncertain (Warner et al., 1997), however,

direct deposition estimates based on data collected in the Elms region of the Chesapeake Bay Atmospheric Deposition Study (the nearest region to the District of Columbia) suggest that direct deposition of iron and zinc may be significant (ICPRB, 1996); direct deposition of PCBs, arsenic, cadmium, chromium, copper, nickel, lead, and selenium were also estimated, but were not as high as for iron and zinc.

The total number of other known and unknown potential point sources of chemical contamination to the Anacostia River may be large, as the following official accounting of known facilities and sites illustrates. The Anacostia River Toxics Management Action Plan reported that, as of 1996, approximately 50 sites in the Anacostia River watershed had been or were being investigated under CERCLA (many of which required no further action or were delisted for brownfield development at that time), while the USDA Beltsville Agricultural Research Center was identified as an NPL site under CERCLA (ICPRB, 1996). Within the District of Columbia, there were 939 RCRIS sites and 6 Toxic Release Inventory facilities reported under EPCRA. None of the 32 CERCLIS sites in the District were on the NPL list, but the U.S. EPA had recently proposed that the Washington Navy Yard be added to the list (McCabe, 1997). McDonald et al. (1994) and McDonald (1998) identified the names, locations, and substances of concern for CERCLIS sites in the District, including those in the Anacostia watershed, as well as the locations of permitted air pollution dischargers, permitted water pollution dischargers, major generators of hazardous waste, and leaking underground storage tanks.

The Warner et al. (1997) study concluded that permitted discharges constitute the majority of known point source discharges to the surface waters of Anacostia River tributaries (and identified the locations of facilities holding active NPDES permits). The study indicated that the permitted discharges were estimated to contribute less than 1% of the total loadings of arsenic, chromium, copper, lead, zinc, and chlordane to the watershed as a whole, relative to nonpoint sources and CSOs. Since not all NPDES permits require reporting loadings of these chemicals, the reported loading estimates were likely to somewhat underestimate the actual combined loadings from all NPDES permit-holders. In general, this study was not sufficiently focused on chemical loadings to identify primary chemical sources. The review considered total loading of all types of pollution, evaluated for only a limited number of individual chemicals, and considered loadings from only three types of sources: nonpoint stormwater runoff, CSOs, and permitted industrial and municipal discharges.

2.2.3 ORGANIZATIONS, PROGRAMS, AND ACTIONS RELATED TO ENVIRONMENTAL MANAGEMENT OF THE ANACOSTIA RIVER WATERSHED

A variety of organizations and governmental environmental offices have contributed to restoring the Anacostia River watershed. As of 1995, hundreds of engineering, ecological restoration, education and outreach projects had been completed in the Anacostia watershed during the previous decade, at a total cost of approximately $130 million (MWCOG, 1995). Other initiatives have been implemented since 1995. The following section provides thumbnail sketches, in alphabetical order, of organizations, programs, and specific actions related to environmental management of Anacostia watershed pollution. These sketches were drawn from information in the documents made available for the screening level risk assessment; this section is not intended to be an exhaustive synthesis of the history of environmental management in the Anacostia River watershed.

Anacostia River Toxics Alliance
The Anacostia River Toxics Alliance, a partnership of a number of public and private members, was formed to address problems related to hazardous chemicals in the Anacostia watershed (AWRC, 1999).

Anacostia Watershed Restoration Committee (AWRC)
The Anacostia Watershed Restoration Strategy Agreement was signed in 1984 by the state of Maryland

and the District of Columbia (MWCOG, 1986), and was expanded to form the Anacostia Watershed Restoration Committee in 1987, including Prince Georges and Montgomery Counties, as well as the US Army Corps of Engineers , the MWCOG (administrator of the agreement) and the Interstate Commission on the Potomac River Basin (ICPRB) (ICPRB, fall 1994, 1996). The four jurisdictions adopted a Six-Point Action Plan in 1991, the first goal of which was to reduce chemical contaminant loads and improve water quality in the tidal Anacostia (ICPRB, fall 1994 ; MWCOG, 1991).

A Memorandum of Understanding was signed in 1996 between the Anacostia Watershed Restoration Committee (AWRC) and U.S. EPA Region III to formally underscore that the two parties will jointly cooperate and coordinate in efforts to restore the Anacostia watershed (AWRC, 1996; U.S. EPA, 1996).

In 1996, the AWRC established the Anacostia Watershed Citizens Advisory Committee (AWCAC) to provide a conduit for input from local residents into the watershed restoration effort (AWRC, 1998). In 1997, the AWCAC co-hosted a watershed-wide cleanup event involving over 800 volunteers and planned to hold town meetings across the watershed in 1998 to gain a better understanding of citizen concerns.

The 1998 Anacostia Watershed Restoration Progress and Conditions Report (1990–1997) was issued by the AWRC to provide an update on the progress made toward attaining each of the six restoration goals, including an assessment of the state of knowledge and actions concerning toxics in the Anacostia (MWCOG, 1998).

Government of the District of Columbia (GDC)

A Kingman Lake water quality model using the U.S. EPA Water Quality Analysis Program (version 5.0; WASP5) was developed as a planning level tool for the District of Columbia Environmental Regulation Administration to address Kingman Lake water quality (particularly algal growth and dissolved oxygen) and to evaluate the impacts of alternative proposed restoration methods (Badruzzaman and Nemura, 1993).

In response to a directive from the Executive Council of the U.S. EPA's Chesapeake Bay Program to develop a regional action plan to address toxic chemical issues in the Anacostia River, the District of Columbia organized two stakeholder meetings in 1995 which focused on scoping the vision, and prioritizing goals and objectives for a Toxics Management Action Plan for the Anacostia River (GDC, 1995).

In a 1995 news release, PEPCO announced an agreement with the District of Columbia concerning participation in Anacostia River restoration, specifically, constructing wetlands, enhancing the Anacostia fishery, shore cleanups, planting trees, and education (PEPCO, 1995).

The Anacostia River Toxics Management Action Plan was prepared by the Interstate Commission on the Potomac River Basin (ICPRB) and released by the District of Columbia in 1996 as part of the District's commitment to the U.S. EPA's Chesapeake Bay Program (ICPRB, 1996). The plan summarized the findings of studies as of 1996 that had evaluated contaminant levels in surface water, sediment, and biota, and addressed five major areas of management to reduce the impact of chemical contaminants on human and ecological health: 1) coordination and funding; 2) public awareness; 3) research and monitoring; 4) source control; and 5) sediment remediation. The initial scope of the plan was confined to the District portion of the Anacostia, however, provisions were made for extending the plan into the upper Anacostia watershed in Maryland if ongoing monitoring of the Anacostia tributaries so indicated.

The District of Columbia has repeatedly recommended that efforts to restore the watershed should be undertaken at the watershed level (GDC, 1994, 1998). The District indicated that control of chemical contaminant inputs from upstream sources in Maryland must be implemented in order for the action plan

to be a success (GDC, 1998).

A *Special Tributary Strategy for Federal Lands in the District of Columbia* is a cooperative effort by Federal facilities within the District of Columbia to control stormwater runoff and reduce nutrient loadings to District waterbodies, including the Anacostia River (GDC, 1998).

Stream bank restoration of Watts Branch was planned to reduce direct sediment and pollutant runoff from impervious surfaces; a "continuous" monitoring program and a USGS gauging station have also been installed for Watts Branch (GDC, 1998).

The District of Columbia provided specific recommendations for point source and non-point source water pollution control programs (GDC, 1998). One recommendation was to "establish a mechanism to reduce human health and environmental risks from groundwater impacted by past activities" (GDC, 1998).

Interstate Commission on the Potomac River Basin (ICPRB)
The Interstate Commission on the Potomac River Basin (ICPRB) initiated *The Potomac River Watershed Visions Project*, which encompasses a large geographic area, including the Anacostia River watershed, and which was designed, in part, to complement the nutrient reduction strategy for the Potomac River basin developed as part of the U.S. EPA Chesapeake Bay Program (Cummins, 1994).

Metropolitan Washington Council of Governments (MWCOG)
MWCOG is an independent, non-profit regional organization comprised of representatives from 17 local governments in the Washington, DC area, as well as members from the Maryland and Virginia legislatures and the U.S. Senate and House of Representatives.

In a 1995 report, a six-part Anacostia Special Study was described that had the goal of developing a Comprehensive Restoration Plan for the Anacostia watershed (MWCOG, 1995). Projects contributing to the Comprehensive Restoration Plan that were related to chemical assessment included an Existing Source Assessment, Long-Term Monitoring Plan, and Water Quality Report.

Maryland-National Capital Park and Planning Commission (M-NCPPC)
As of 1992, watershed development was reported to have progressed for 28 years according to a plan developed by the M-NCPPC to direct growth of commercial, industrial, and heavy residential land uses to transportation corridors, and maintain low intensity use areas (such as parks, recreation centers, reserved open spaces, and low density residential housing) as buffer zones between the heavy use areas. In addition, parklands were maintained to buffer streams in the watershed from heavily urbanized, high-intensity use areas (U.S. EPA-CBP, 1992).

United States Army Corps of Engineers (USACE)
Since 1961, the Anacostia River has been periodically dredged by the USACE, and dredged materials have been used to build Children's Island, the National Arboretum, the Kenilworth Aquatic Gardens, and riverside parkland (GDC, 1994). Dredging from the Conrail Bridge to the Bladensburg Marina was implemented in 1992, and dredged materials from this effort were used to restore Kenilworth Marsh tidal emergent wetlands. It has been hypothesized that dredging may have diluted the concentration of contaminants in the surface sediment by introducing significant quantities of *cleaner* sediments from dredge depths of up to six feet (GDC, 1994).

Kenilworth Marsh restoration was linked to a nearby dredging operation in the Anacostia River by the USACE and was completed in 1993, doubling the size of the original planned restoration area (AWRC, 1998). Collaborative efforts were planned between the District of Columbia and the USACE to develop further wetlands in the Kingman Lake area and riparian wetlands on the Anacostia (GDC, 1994, 1998).

In accordance with the 1994 Agreement of Federal Agencies on Ecosystem Management in the Chesapeake Bay, the USACE developed a Biennial Federal Work Plan for the Anacostia River Watershed in 1996 that identified federal agencies with facilities in the watershed, outlined the roles that the agencies were currently taking toward achieving the goals of the AWRC six-point action plan for the watershed, and suggested further assistance that they could provide (USACE, 1996).

United States Environmental Protection Agency - Region III

An *Inventory of EPA Region III Activities/Initiatives in Support of Anacostia Improvement Goals* was compiled in 1994 that included several activities regarding chemical issues in the watershed, including site inspections of potential sources of chemical contamination in the watershed (U.S. EPA [Initiatives], 1997).

The U.S. EPA Region III Anacostia Ecosystem Initiative was initiated in 1994 to work toward attaining the following six objectives: target existing U.S. EPA programs in the watershed; control combined sewer overflows and stormwater; public education and outreach; build federal sector support for watershed restoration; reduce environmental and human health risks; and support community-based environmental justice initiatives (U.S. EPA, 1998).

A 1998 update report on the U.S. EPA Region III Anacostia Ecosystem Initiative indicated that the U.S. EPA had required removal of the sediment from sewers in the Southeast Federal Center as part of a NPDES permit issued to the General Services Administration because of high levels of PCBs and heavy metals found in the sewers by the U.S. EPA (U.S. EPA, 1998). The report also indicated that the U.S. EPA proposed that the Washington Navy Yard become an NPL site, and that the Navy had been ordered to begin cleanup of contaminated soil, surface water, and groundwater at the Naval Surface Warfare Center on Paint Branch in the Anacostia watershed.

2.2.4 PROGRAMS AND STUDIES INITIATED TO GATHER DATA CONCERNING CHEMICAL STRESSORS IN THE ANACOSTIA WATERSHED.

A comprehensive review of studies conducted to measure chemical contamination levels in various tidal Anacostia media is provided in the problem assessment section of *The Anacostia River Toxics Management Action Plan* (ICPRB, 1996). Some of these studies were available for the screening level risk assessment. The following are brief summaries of studies and sampling programs described in reports that were available for the screening level risk assessment and that have contributed to the overall information base concerning chemical contamination levels and toxic effects to biota in the Anacostia watershed. The studies are organized alphabetically by the organization or program that either sponsored or conducted the study.

East Station Project.

Biota, Soil, and Groundwater. Habitat quality and benthic community structure were evaluated at six sampling locations in 1988 in the lower Anacostia in the vicinity of the 12th Street Bridge and the Navy Yard as a part of the East Station Contamination Study (Hydro-Terra, Inc., 1989); the study concluded that the area was significantly impacted. An attachment to the Hydro-Terra, Inc. (1989) report indicated that PAHs and PCBs were detected in the surface soil between the 12th Street perimeter fence and a retaining wall below, identified elevated sediment PAH concentrations related to a groundwater contamination plume in the fill behind the seawall that was being monitored by on-shore shallow wells, and identified low concentrations of benzene, toluene, ethylbenzene and xylenes in the water collected from the 12th Street sewer, but concluded that the sewer contamination probably did not significantly impact Anacostia River water quality.

Surface Water and Sediment. Hydro-Terra, Inc. (1997) reported analytical data, but no interpretation, of a 1996 surface water and sediment sampling effort just north of the 12th Street Bridge on the west shore of the Anacostia River. Data from this study are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* .

Government of the District of Columbia (GDC)

Surface Water and Biota. The District of Columbia's Water Management Division of the Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, has prepared a series of reports to the U.S. EPA and the U.S. Congress concerning water quality information for the District of Columbia's surface and ground waters as required under Section 305(b) of the federal Clean Water Act (GDC, 1994, 1996, 1998).

In 1997, an existing fixed station water monitoring program (for evaluating physical/chemical water quality parameters, heavy metals, and pathogen analysis) was revised to include 12 Anacostia River stations, 3 stations in Watts Branch, 2 stations in Kingman Lake, and 1 station in Kenilworth Marsh (GDC, 1998). In addition, the District developed a tributary monitoring program in 1995 for monitoring smaller tributaries for periodic standard water quality parameters and biological/habitat assessments. In 1997, the tributary monitoring program was implemented to periodically gather data concerning stream hydrology, morphological conditions, and types of organisms living in the streams.

The Steuart Petroleum Company, in cooperation with the District of Columbia and the National Park Service, developed a monitoring study to evaluate the residual effects of an oil release that occurred in 1992 into the Anacostia River (Pinkney et al., 1993). Based on compositional analysis, they concluded that the spilled fuel oil was not a significant source of total petroleum hydrocarbons and/or PAHs that were detected in surface water, sediment, and fish nearby in the Anacostia River (Pinkney et al., 1993).

Groundwater. Approximately 325 sites have been identified in the District of Columbia as having confirmed groundwater contamination (GDC, 1998); the proportion that lie within the Anacostia watershed and the identity of the chemical contaminants were not reported. Sources and sites of groundwater contamination in District of Columbia waters were under investigation as of 1994, and were to be compiled into the *District of Columbia's Sources of Potential Ground Water Contamination Inventory* (GDC, 1994).

The District of Columbia conducted a study of contaminant levels in groundwater samples collected during a single sampling event in July, 1996, from one monitoring well in each of the two Kenilworth Landfills on opposite banks of the Anacostia (Montaser, 1997). One of the landfills was capped with soil and is now the Kenilworth Park. The report indicated that low levels of cadmium, copper, iron, lead, and zinc were detected in one or both wells, and that no pesticides, PCBs, or mercury were detected in the sampled groundwater.

Biota. A series of studies was supported by the District of Columbia and the National Park Service to investigate benthic macroinvertebrates in the Anacostia River. In 1984 survey, no live macrobenthic animals of any type were found upriver of the Pennsylvania Avenue bridge in the tidal Anacostia (Phelps, 1985). A related study reported reduced gonadal mass and severely impeded spawning in clams introduced to the lower Anacostia in an *in situ* bioassay conducted in 1986 (Phelps, no date), but a study on the toxic effects of Kenilworth Marsh sediments to clams was inconclusive (Phelps and Clark, 1988).

In the early 1990s, the District of Columbia's Water Hygiene Branch developed a program to monitor the health of District streams using rapid bioassessment of habitat quality and biological water quality, including 11 sites within the Anacostia River watershed (Banta and Horowitz, 1992 ; Banta, 1993).

Interstate Commission on the Potomac River Basin (ICPRB)

Surface Water. A 1990 report from the ICPRB presented data from two sampling locations in the Anacostia River in a 1986–1987 water quality survey of standard physical/chemical water quality parameters and several heavy metals (Haywood and Focazio, 1990); the report did not provide an interpretation of the results.

A year-long surface water monitoring project was undertaken during 1995–1996 by the ICPRB to evaluate chemical loadings from the Northeast and Northwest Branches to the tidal Anacostia River (Gruessner et al., 1997). Surface water samples and concurrent flow data were collected throughout the year under both storm and non-storm conditions. Various metals, PCBs, PAHs, and organochlorine pesticides were detected in the particulate phase of the samples, and the report concluded that the large amount of suspended matter transported into the tidal Anacostia from both branches may constitute a significant transport mechanism for these chemical contaminants from upstream sources to the tidal Anacostia.

Sediment and Biota. In an October 1989 study performed for the ICPRB, Limno-Tech, Inc. (LTI, 1990) sampled surface sediments (top six inches) throughout the District of Columbia area of the Potomac River Basin, including throughout the tidal Anacostia, Kingman Lake, Watts Branch, and Hickey Run. Samples were analyzed for U.S. EPA's list of priority pollutants and detections for over 60 chemicals on the list were reported. The highest contaminant levels and the greatest numbers of detected priority pollutants were found between the Benning Road Bridge and the South Capital Street Bridge of the Anacostia River. Contaminant concentrations were compared to U.S. EPA Great Lakes Sediment Guidelines, and the greatest frequencies of exceedance in the tidal Anacostia River were reported to be in the organochlorine pesticide/PCB and metals classes of chemicals (LTI, 1990).

During the 1990s, the ICPRB, in cooperation with the District of Columbia and other interested governmental offices and private organizations, conducted a series of studies and prepared several reports concerning chemical contaminants in fish and sediments in District waters, including the tidal Anacostia River.

In June, 1991, sediments were sampled throughout the major District of Columbia waterways, including six stations in the lower Anacostia River south of the Pennsylvania Avenue Bridge, and five stations throughout Kingman Lake, and then analyzed for levels of PAHs, PCBs, pesticides, and metals (Velinsky et al., 1992). Some of the sampling stations were situated in the river proximal to sewer outfalls, and sediment samples were also taken at the outfalls and further up inside the sewers themselves to ascertain whether sewers were sources of river sediment contamination. Lead, cadmium, mercury, PAHs, PCBs, and total DDT were detected in several places, such as near the Washington Navy Yard. Sediment toxicity tests in amphipods and macroinvertebrate community analysis also indicated a severe degree of biological impairment in the lower Anacostia River. The study concluded that sewers were a major source of chemical contamination in the Anacostia River, and that the lower Anacostia River was the area of greatest concern within the waters of the District of Columbia. The study also provided a history of other earlier sampling efforts to evaluate the nature and extent of chemical contamination in several media in District of Columbia waters. Data from the Velinsky et al. (1992) study are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* .

In a re-analysis of the Velinsky et al. (1992) data, Wade et al. (1994) and Velinsky et al. (1994) concluded that decreasing concentration gradients between sewer, outfall, and river sediment samples, particularly near the Washington Navy Yard, suggest that urban runoff may be a major non-point source of metals, hydrocarbon (e.g., PAHs), PCB, and DDT contamination to Anacostia River sediments. They also reported that for certain contaminants, like PAHs, the outfall sediment concentrations indicate diffuse

distributions, while for other contaminants such as PCBs, the distributions suggest that specific outfalls may be major contributors (Wade et al., 1994).

A 1994 study conducted by the District of Columbia and reported by the ICPRB evaluated 129 U.S. EPA priority pollutants as well as separate analyses for PCBs and PAHs in tissues of fish collected from 1989 to 1992 throughout the major District of Columbia waterways, including 18 samples collected in the tidal Anacostia river below the New York Avenue bridge (Cummins and Velinsky, 1993 ; Velinsky and Cummins, 1994). Metals detected most often in the edible portions of the fish were arsenic, selenium, and mercury, while total PCBs, organochlorine pesticides, and certain PAHs were found in highest concentrations in the American eel and channel catfish. A potential for an excess cancer risk greater than 10^{-4} to 10^{-3} from consumption of total PCBs or chlordane in fish was estimated. FDA action levels for total PCBs and chlordane were exceeded in 4 and 1 fish samples, respectively, that were collected from District waters.

A follow-up 1996 study by the District of Columbia and ICPRB evaluated 129 U.S. EPA priority pollutants as in addition to PCBs and PAHs in tissues of fish collected throughout the major District of Columbia waterways, including from the upper tidal Anacostia River (New York Avenue Bridge to the CSX railroad bridge near the south end of Kingman Lake) in 1994, from Kenilworth Marsh in 1993, and from the lower tidal Anacostia River (the CSX railroad bridge near the south end of Kingman Lake to the confluence with the Potomac River) in 1993 (Velinsky and Cummins, 1996). Samples were composited apparently by sampling location, sampling event, and species. Detectable levels of many chemicals were in the edible portions of the sampled fish, with the highest levels of metals found in predatory fish such as the sunfish and largemouth bass. The highest levels of bioaccumulative organics (such as PCBs, PAHs, and chlordane) were found in the channel catfish and common carp from the lower Anacostia. The study concluded that levels of certain contaminants in fish may pose a health risk to people who consume the fish. Data from the Cummins and Velinsky (1993) and Velinsky and Cummins (1994, 1996) studies are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* . Velinsky and Cummins, (1994, 1996) regarded the data from both studies as inadequate for detecting geographic or temporal trends in contaminant levels in fish.

Metropolitan Washington Council of Governments (MWCOG)

Surface Water and Biota. A Coordinated Anacostia Monitoring Program (CAMP) was organized in 1984 between the District of Columbia, the state of Maryland, Prince Georges County, and Montgomery County and other interested governmental offices to provide for bimonthly, same-day surface water sampling at a network of 46 sites throughout the Anacostia watershed, most of which are in the tidal Anacostia River (U.S. EPA-CBP, 1992). The District of Columbia maintained 9 other water quality stations as of 1992, in addition to those monitoring stations maintained for CAMP. The MWCOG coordinates sampling activities, compiles data from the CAMP into computerized databases, and develops annual reports on water quality condition in the watershed (U.S. EPA-CBP, 1992).

In 1986, the water quality data from sampling locations throughout the Anacostia River watershed were compiled from records of sampling events from the early 1980s provided by Montgomery County, Prince Georges County, District of Columbia, and the state of Maryland (MWCOG, 1986). In addition to standard general water quality parameters and assessments of biological resources, data concerning concentrations of metals and pesticides in surface water and in fish were summarized. Some information regarding dredging events by the USACE was also provided.

A 1994 Anacostia Watershed Water Quality Report on conditions in the watershed during 1987–1990 was produced by the MWCOG as part of the broader Comprehensive Restoration Plan and provided standard water quality and biological assessment data in the tidal river, tidal tributaries, and upper tributaries (Herson-Jones et al., 1994). The investigators divided the tidal Anacostia into segments based on

physiography and locations of tributary and CSO inputs to the system. In a spatial re-analysis of Velinsky et al. (1992) sediment contamination data of the tidal Anacostia below Kingman Lake, Herson-Jones et al. (1994) found a peak in concentrations of cadmium, mercury, lead, zinc, DDT, and PCBs, immediately downstream of the Navy Yard . The report identified the Navy Yard, the Bureau of Engraving and Printing, the old Lionel freight yard and the U.S. Botanical Gardens as potential sources of the down river contamination; whereas, the spatial profile for chlordane was attributed to a possible source within or upstream of Kingman Lake.

In recognition of repeated oil releases to Hickey Run, the MWCOG initiated a comprehensive pollution abatement study in 1993, which included a detailed map of storm drains in the catchment area and plan to develop a hydrocarbon spill storm drain tracing system to identify the locations of petroleum sources (Shepp and Cole, 1993 ; Shepp and Parikh, 1995). The report indicated that previous investigations had identified METRO, AMTRAK and the Greyhound Bus Company as likely contributors based on oil fingerprinting but that there were also a large number of smaller potential contributors (GDC, 1998 ; Shepp and Parikh, 1995).

PEPCO

Surface Water. In an experiment conducted from 1995–1997 during a PEPCO construction project, concentrations of certain contaminants, including chlordane and some metals, appeared to be elevated in the water column during and after dredging operations compared to pre-dredging levels, but dredging did not appear to affect concentrations of PCBs, chlordane, or metals (the only analytes tested) in the sediment (Loos, 1999). Data from the Loos (1999) study are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* .

Prince Georges County

Surface Water and Biota. A comprehensive biological assessment was conducted in the spring of 1999 in streams and watersheds of Prince Georges County, MD, including those in the Anacostia River watershed, as the initial event in a 5-year periodical assessment plan (Stribling et al., 1999). The assessment rated 11 of the 12 randomly selected sites in the Anacostia River watershed as poor or very poor regarding the capacity to support aquatic life, and all three targeted sites in Lower Beaverdam Creek were rated as very poor (using ratings of good, fair, poor, and very poor).

United States Army Corps of Engineers (USACE)

Sediment. The USACE, Baltimore District, sponsored a study of physical and chemical analyses of sediment collected at 3 depths (0–1, 1–5, and 5–9 feet) from four sampling locations in the Anacostia River in the river reach that parallels Kingman Lake (Groundwater and Environmental Services, 1998). The study identified numerous semivolatiles, pesticides/PCBs, and metals in sediments that were characterized as dark gray-brown silt with some fine-grained sand and little clay.

United States Environmental Protection Agency

Surface Water. In 1997, the U.S. EPA conducted a comprehensive *Environmental Characterization of the District of Columbia* that reviewed and summarized existing information from both human health and ecological perspectives (McCabe, 1997). The report characterizes sources of pollution to air and surface water. Data gaps in the current knowledge concerning human health issues related to exposures to toxic chemicals were identified by the U.S. EPA, and included the need for a regional perspective on the dynamics of pollutant migration from surrounding counties and the District, and the need for data on human activity patterns including fishing, swimming, and wading.

U.S. EPA Region III funded a study of the effects of stormwater runoff on the water quality of the Anacostia River, which concluded that the non-tidal watershed contributes significant loadings of chemical contaminants to the upper tidal Anacostia waters within 24 hours of significant rainfalls, while

urban runoff is the primary source of contaminants in the lower tidal Anacostia River (Velinsky et al., 1999). Data from the Velinsky et al. (1999) study are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* .

Sediment. In 1995, as part of the overall U.S. EPA Region III Anacostia River Initiative, the Annapolis Operations Section of Region III performed a special sampling investigation of contaminant levels in residue/sediment within the major storm sewer systems that drain the Washington Navy Yard (Clark and Crutchley, 1995). Heavy metals (particularly zinc, lead, copper, nickel, chromium, and mercury) and PCBs were detected in solid residues collected from storm sewers that drain the Washington Navy Yard, and heavy metals were detected in one adjacent District of Columbia municipal storm sewer. The report concluded that it was not possible to tell whether contaminants were deposited from ongoing contaminant releases or from historical releases, and advised that the Navy Yard should vacuum out the solids currently in the sewers and monitor new deposits for contaminants to see whether release is on-going. The report indicated that specific Navy Yard buildings were identified that are good candidates for further PCB source investigation.

Clark and Gower (1995) compared the findings of Clark and Crutchley (1995), who sampled deposits within sewers that drain the WNY, to those of Velinsky et al. (1992), who sampled sediments in the riverbed in the lowermost 4-mile reach of the tidal Anacostia River. A sample that had a PCB concentration of 227 ppm was obtained from one of the Navy Yard's storm sewers. However, neither the Navy Yard nor the adjacent Southeast Federal Center reported any PCB transformers or other electrical equipment containing PCBs. Several Anacostia River bottom sediment samples showed PCB concentrations greater than the U.S. EPA Sediment Quality Guideline for PCBs; the furthest upstream exceedance occurred at the Pennsylvania Avenue Bridge. Based on a comparison of sediment contaminant levels to the respective U.S. EPA sediment quality guidelines, Clark and Gower (1995) concluded that the contaminants of greatest concern in the lower Anacostia are lead, nickel, silver, zinc, and PCBs, with exceedances ranging from 2 to 5 times the guideline. They also concluded that it was unclear whether PCB contamination was from past releases or from continued stormwater runoff.

United States Fish and Wildlife Service (USFWS)

Sediment and Biota. Surveys conducted in 1992 and 1996 found that liver and integumentary tumors were prevalent in brown bullhead populations of the Anacostia River in 55% and 23% of fish sampled, respectively (Harshberger et al., 1997 ; Pinkney, 1999). The Pinkney (1999) report noted that the prevalence of these tumors in Anacostia River fish was comparable to those observed at sites in the Great Lakes region; a follow-up analysis was underway to examine possible associations between tumor prevalence and chemical contamination. An earlier study in 1987 surveyed contaminant concentrations and incidence of lesions in channel catfish and largemouth bass in District of Columbia waters, including the tidal Anacostia River; the highest concentrations of chlordane and DDT were obtained in the lower Anacostia, and the incidence of total gross lesions and non-parasitic lesions was statistically significantly greater at two sampling sites in the Anacostia compared to a reference site located at Fletcher's Boat House on the Potomac River, upstream of the Anacostia River confluence (Block, 1990). Data from the Block (1990) study are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* .

Results from a 96-hour pore water bioassay and a 10-day sediment bioassay indicated that sediment sampled in August of 1993 from Kingman Lake and from the Anacostia River near Kingman Lake was not acutely toxic to the amphipod *Hyalella azteca* (USFWS, 1994, 1997).

Dredged Anacostia River sediment that was used to restore Kenilworth Marsh was sampled shortly after placement at the marsh in 1993, and both sediment solids and sediment pore water were evaluated for contaminant concentrations; analytical data were provided in a 1997 data report (USFWS, 1997). A 1998 report prepared by the U.S. Fish and Wildlife Service found elevated levels of chromium, copper,

lead, nickel, total PCBs, total DDT, and total chlordane in sediments, killifish, and/or cattails in Kenilworth Marsh compared to a regional reference location and to national averages, and indicated that the observed levels were associated with adverse effects in fish-eating wildlife (Murphy et al., 1998). The report attributed the contamination to the use of dredged Anacostia River sediments that were used restore the marsh, and recommended that future use of dredge sediments from the Anacostia for wetland restoration should be evaluated on a case-by-case basis (Murphy et al., 1998). Data from both the USFWS (1997) and Murphy et al. (1998) studies are included in the database used for the screening level human health risk assessment; see Section 5 - *Existing Data Summary/Compilation* .

United States Navy

Surface Water and Sediment. A Navy Research Program entitled *Contaminant Distribution and Fate in Anacostia River Sediment* was funded by the Environmental Regulatory Coordinator of the Washington Naval Base and performed by the Naval Research Laboratory. A particulate transport survey that evaluated sediment transport mechanisms within the tidal Anacostia River (Coffin et al., 1998) and an extension study concerning the fate and transport of PAHs and metals in the tidal Anacostia (Coffin et al., 1999) were funded under this program.

Coffin et al. (1998, 1999) attributed thermal stratification observed during June 1997 through February 1998 in the center of the lower Anacostia River (between the Washington Naval Yard and the Pennsylvania Avenue Bridge) to tidal circulation, which may periodically move cooler Potomac River water into the lower surface water stratum of the lower Anacostia River. Stratification was observed to be greatest during flooding tides at these locations. However, no thermal stratification was seen either at the mouth of the Anacostia River or upstream of the Pennsylvania Avenue Bridge. The hypothesis that tidal circulation may resuspend and transport Potomac River sediments that are proximal to the Anacostia River mouth into the lower Anacostia River has not been studied. However, Coffin et al. (1999) hypothesized that temperature stratification in the lower Anacostia River may indicate tidal upflow of colder Potomac River water, which may increase upriver transport of TSS. Coffin et al. (1999) observed that flow direction at the Pennsylvania Avenue Bridge and in front of the Washington Navy Yard was dependent on the tidal cycle, based on four 3-day measurement events during the period between June 1997 and June 1998.

Coffin et al. (1999) observed both geographical and seasonal variation occurred in TSS in the Anacostia River during the period June 1997 through May 1998. TSS in the tidal Anacostia River increased with distance from the confluence of the Potomac River at sampling locations up to the Bladensburg Marina, and TSS concentrations were most consistently low in November and consistently high in February, while TSS in May and June showed relatively high variability. Coffin et al. (1999) suggested that relatively low TSS concentrations at the Washington Navy Yard may be due to greater sedimentation occurring at that location, due to lower current velocities. They hypothesized that the upper tidal region of the Anacostia was a source of TSS-associated PAHs to the lower Anacostia, based on observations of higher TSS levels in the upper tidal Anacostia than in lower reaches.

Coffin et al. (1999) hypothesized that reduced current velocity in front of the Washington Navy Yard may indicate that enhanced sediment deposition could occur at that point, based on the observation that mean current velocity at the Pennsylvania Avenue Bridge, as measured using Acoustic Doppler Current Profiles (ADCP), was consistently greater at several measurement depths than in front of the Navy Yard. Measurements using a hand-held velocity meter (calibrated to the ADCP readings) also indicated statistically significantly reduced mean current velocities in the Anacostia River in front of the Washington Navy Yard over the period June 1997 through February 1998, compared to readings obtained at the confluence with the Potomac River, at the Pennsylvania Avenue Bridge, and at the Benning Road Bridge (Coffin et al., 1999). Sediment deposition measured by sediment traps at the Washington Navy Yard was reportedly similar to values from the upper tidal Anacostia, although supporting data were not

provided (Coffin et al., 1999).

Coffin et al. (1999) also hypothesized that temperature stratification observed in the lower Anacostia River from June 1997 through February 1998 may indicate tidal upflow of colder Potomac River water, which may in turn affect TSS deposition in certain (unspecified) areas.

PAH concentration in solids obtained in sediment traps near the Washington Navy Yard was greater than or equivalent to concentrations either at the confluence with the Potomac River, or PAH concentrations measured in deposited solids at locations upstream of the Navy Yard (Coffin et al., 1999).

Coffin et al. (1999) recommended dye tracer studies to analyze tidal excursions in various segments of the river, Beryllium tracer studies to determine sedimentation rates over the short term, microbial degradation studies of PAHs, immunological toxicity assays of PAHs in the river, and stable carbon isotope analyses to fingerprint PAH sources throughout the tidal and nontidal Anacostia River.

2.2.5 ITEMIZED SUMMARY OF INFORMATION ON POTENTIAL SOURCES OF CHEMICAL STRESSORS IN THE ANACOSTIA WATERSHED AND DATA GAPS RELEVANT TO PREDICTING RISK FROM CHEMICAL EXPOSURES IN THE TIDAL ANACOSTIA RIVER

Potential Sources. Identifying point sources of chemical contamination and the nature and magnitude of chemical releases from those sources is informative for estimating current and potential future human exposures, and is necessary for remediation planning if unacceptable risks are associated with the predicted exposures. However, the identity of a point source from the perspective of exposure modeling may differ from the corresponding point source considered for remediation. For instance, a specific stormwater sewer or CSO outfall, or tributary confluence, may constitute a point source of chemical contaminants to the tidal Anacostia (e.g., Velinsky et al., 1992, 1994 ; Wade et al., 1994), when considered from the perspective of modeling contaminant transport within the tidal river proper. Whereas, identifying the specific location of the ultimate source of the PCBs within the sewer or tributary's drainage area would be important from the perspective of remediation. Information concerning sources of chemical contamination that was collected from documents available for the screening risk assessment is presented below.

X Current point sources of ongoing chemical release to the tidal river may include pockets of contamination in groundwater, soil, or other below-ground sources such as underground storage tanks or landfill material, that may have been deposited by past activities. Certain documents that were available for the screening risk assessment identify some of the potential sources of groundwater contamination (GDC, 1994, 1996, 1998).

X Approximately 325 sites have been identified in the District of Columbia as having confirmed groundwater contamination (GDC, 1998); the proportion that lie within the Anacostia watershed was not reported. Sources and sites of groundwater contamination in District of Columbia waters were under investigation as of 1994, and were to be compiled into the *District of Columbia's Sources of Potential Ground Water Contamination Inventory* (GDC, 1994). Some work has been initiated to study groundwater dynamics in the District of Columbia (GDC, 1994). A comprehensive District of Columbia *Groundwater Resource Assessment Study* was prepared in 1994, but was not available for the screening risk assessment. Available information indicates that groundwater generally flows toward the surface water bodies in the Anacostia watershed, except in the lower reaches of the tidal Anacostia, where tidal action causes an interchange of groundwater and surface water in both directions (GDC, 1994). The adequacy of the reported data for constructing a groundwater-surface water hydrodynamic model of the tidal Anacostia could not be evaluated since the studies themselves were not available for the screening risk assessment.

X McCabe (1997) identified 7 facilities in the District that had active permits, as of 1997, to discharge wastewater directly into the surface waters of the tidal Anacostia. Chemicals permitted for discharge at these facilities included oil and grease, chromium, zinc, copper, lead, cadmium, silver, and mercury. The identity and locations of facilities with NPDES permits throughout the Anacostia watershed are provided in Warner et al. (1997).

X The total number of other known and unknown potential point sources of chemical contamination to the Anacostia River may be large, as the following official accounting of known facilities and sites illustrates. The Anacostia River Toxics Management Action Plan reported that, as of 1996, approximately 50 sites in the Anacostia River watershed had been or were being investigated under CERCLA (many of which required no further action or were delisted for brownfield development at that time), while the USDA Beltsville Agricultural Research Center was identified as an NPL site under CERCLA (ICPRB, 1996). Within the District of Columbia, there were 939 RCRIS sites and 6 Toxic Release Inventory facilities reported under EPCRA. None of the 32 CERCLIS sites in the District were on the NPL list, but the U.S. EPA had recently proposed that the Washington Navy Yard be added to the list (McCabe, 1997). McDonald et al. (1994) and McDonald (1998) identified the names, locations, and substances of concern for CERCLIS sites in the District, including those in the Anacostia watershed, as well as the locations of permitted air pollution dischargers, permitted water pollution dischargers, major generators of hazardous waste, and leaking underground storage tanks.

X Annual loadings of selected metals, PAHs, pesticides, and PCBs (both particulate and dissolved) from the Northeast and Northwest Branches to the tidal Anacostia river were estimated using mean measured concentrations (measured during both storm and non-storm conditions) and mean annual flow rates from USGS stations (Gruessner et al., 1997).

X A comprehensive study of chemical contaminant sources throughout the Anacostia watershed prepared by the District of Columbia identified nonpoint sources as the primary concern, although the study acknowledged that no information concerning groundwater contributions was used in the evaluation (Warner et al., 1997). With respect to chemical contaminants in the Anacostia watershed, the source report addressed loadings of petroleum hydrocarbons, lead, and zinc, in particular. Hickey Run was reported to have a history of episodic petroleum hydrocarbon inputs. A plan to trace the specific point sources in Hickey Run is being implemented by MWCOG (Warner et al., 1997). Eighty-five percent of the annual loadings of lead were reported to be from CSOs in the tidal Anacostia, while zinc loadings were predominantly from nonpoint sources (Warner et al., 1997). The loadings of arsenic, chromium, and copper were also attributed to nonpoint sources, although the study reported that no data concerning CSO loadings were available for these metals; no other chemicals were evaluated in the source study (Warner et al., 1997). Warner et al. (1997) concluded that permitted discharges constitute the majority of known point source discharges to the surface waters of Anacostia River tributaries (and identified the locations of facilities holding active NPDES permits), but that the permitted discharges were estimated to contribute less than 1% of the total loadings to the watershed as a whole, relative to nonpoint sources and CSOs

X The Interstate Commission on the Potomac River Basin (ICPRB) identified surface runoff, storm and combined sewer discharges, and direct atmospheric deposition as potential sources of hazardous substances detected in sediment sampled in the tidal Anacostia River (Gruessner et al., 1997).

X Velinsky et al. (1992) concluded that sewers were a major source of chemical contamination in the Anacostia River, and that the lower Anacostia River was the area of greatest concern within the

waters of the District of Columbia. The study also provided a history of other earlier sampling efforts to evaluate the nature and extent of chemical contamination in several media in District of Columbia waters. Several documents identify locations of stormwater sewer outfalls and CSOs in the lower tidal Anacostia River (Clark and Crutchley, 1995 ; Clark and Gower, 1995 ; Velinsky et al., 1992, 1994 ; Wade et al., 1994).

X In a spatial re-analysis of Velinsky et al. (1992) sediment contamination data of the tidal Anacostia below Kingman Lake, Herson-Jones et al. (1994) found a peak in concentrations of cadmium, mercury, lead, zinc, DDT, and PCBs, immediately downstream of the Navy Yard . The report identified the Navy Yard, the Bureau of Engraving and Printing, the old Lionel freight yard and the U.S. Botanical Gardens as potential sources of the down river contamination; whereas, the spatial profile for chlordane was attributed to a possible source within or upstream of Kingman Lake.

X Repeated oil releases have occurred to Hickey Run, according to the MWCOG, which initiated a comprehensive pollution abatement study in 1993. The study included a detailed map of storm drains in the catchment area and plan to develop a hydrocarbon spill storm drain tracing system to identify the locations of petroleum sources (Shepp and Cole, 1993 ; Shepp and Parikh, 1995). The report indicated that previous investigations had identified METRO, AMTRAK and the Greyhound Bus Company as likely contributors based on oil fingerprinting but that there were also a large number of smaller potential contributors (GDC, 1998 ; Shepp and Parikh, 1995).

Data Gaps. In general, data concerning chemical loadings from sources, chemical concentrations in various media, chemical transformation, and chemical transport processes have been collected over the course of several decades and throughout the Anacostia River watershed, and as such comprise a spatio-temporal patchwork picture of chemical contamination in the tidal Anacostia. In addition, analytical methods and target analytes differed between studies. However, the tidal Anacostia is a complex, dynamic system. Data have not been collected and analyzed in a coordinated manner in order to develop a comprehensive understanding of chemical contamination sufficient for quantifying current human exposures to various media, or for predicting future human exposures or future contamination conditions under various remediation scenarios. Some specific data gaps are discussed in Section 8.

REFERENCES FOR CHAPTER 2

ATSDR (Agency for Toxic Substances and Disease Registry). 1998. Health Consultation. Anacostia River Initiative, Washington, District of Columbia.

Anacostia Watershed Restoration Committee. 1998. Anacostia Currents. Anacostia Watershed Restoration Committee, Washington, DC. 11 pages.

Anacostia Watershed Restoration Committee and United States Environmental Protection Agency. 1996. Memorandum of Understanding to clarify the mutual commitment and cooperation, as well as enhanced coordination and communication efforts designed to restore and protect the Anacostia Watershed and its tributaries.

Anacostia Watershed Restoration Committee. 1999. Meeting October 5, 1999.

Badruzzaman, A.B.M. and A.D. Nemura. 1993. Kingman Lake Water Quality Model. Metropolitan Washington Council of Governments, Environmental Regulation Administration, Department of Consumer and Regulatory Affairs, Washington, DC. 39 pages + 4 appendices.

Banta, W.C. 1993. Biological water quality of the surface tributary streams of the District of Columbia. American University, Washington, DC. 335 pages.

Banta, W.C. and C. Horowitz. 1992. Rapid bioassessment of macroinvertebrates in select lotic waters of the District of Columbia. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Hygiene Branch, Washington, DC. 45 pages.

Block, E. 1990. Organochlorine residues and histopathological examination of fish from the Potomac and Anacostia Rivers, Washington, DC. U.S. Fish and Wildlife Service, Environmental Contaminants Division, Annapolis, MD. AFO-C90-01 30 pages.

Clark, L.J. and M. Gower. 1995. A brief review and analysis of heavy metals and PCB data lower Anacostia River. U.S. EPA, Region III, Philadelphia, PA. 30 pages.

Clark, L.J. and G. Crutchley. 1995. Special sampling investigation Washington Navy Yard and Environs April 24-27, 1995. U.S. EPA, Region III, Philadelphia, PA. 30 pages.

Clean Water Action Plan Technical Workgroup. 1998. Maryland clean water action plan. Draft 1998 report on Unified Watershed Assessment, Watershed Prioritization and Plans for Restoration Action Strategies. Clean Water Action Plan Technical Workgroup, Maryland. 46 pages.

Coffin, R.B., M. Orr, E. Carey, L. Cifuenties and J. Pohlman. 1998. Contaminant distribution and fate in Anacostia River sediments: Particulate transport survey. Naval Research Laboratory, Washington, DC. 27 pages.

Coffin, R.B., J.W. Pohlman and C.S. Mitchell. 1999. Fate and transportation of PAH and metal contaminants in the Anacostia River tidal region. Naval Research Laboratory, Washington, DC. 17 pages.

Cummins, J.D. 1994. The Potomac River watershed visions project. Interstate Commission on the

Potomac River Basin, Rockville, MD. 50 pages.

Cummins, J.D. and D.J. Velinsky. 1993. 1992 D.C. fish tissue analysis for the evaluation of human health risks. District of Columbia, Department of Consumer and Regulatory Affairs, Water Quality Control Branch, Water Resources Management Division, Washington, D.C. 7 pages.

Government of the District of Columbia. 1994. The District of Columbia water quality assessment 1994 report to the Environmental Protection Agency and U.S. Congress pursuant to Section 305(b) Clean Water Act (P.L. 97-117). Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC. 1200 pages.

Government of the District of Columbia. 1995a. Anacostia River Toxics Management Action Plan. Government of the District of Columbia, Department of Consumer & Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC. 8 pages.

Government of the District of Columbia. 1995b. List of projects for EPA interns.

Government of the District of Columbia. 1996. The District of Columbia water quality assessment 1996 report to the Environmental Protection Agency and U.S. Congress pursuant to Section 305(b) Clean Water Act (P.L. 97-117). Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC. 1200 pages.

Government of the District of Columbia. 1998a. The Abridged District of Columbia water quality assessment 1998 report to the Environmental Protection Agency and U.S. Congress pursuant to Section 305(b) Clean Water Act (P.L. 97-117). Government of the District of Columbia, Department of Health, Environmental Health Administration, Water Quality Division, Washington, DC. 1200 pages.

Groundwater & Environmental Services. 1998. Sediment sampling for chemical and particle size analysis at Anacostia River federal Navigation Channel, Columbia Island Marina, Washington Sailing Marina. U.S. Army Corps of Engineers, Baltimore District CENAB-PL. 146 pages.

Gruessner, B., D.J. Velinsky, G. Foster, R. Mason and J. Scudlark. 1997. Dissolved and particulate transport of chemical contaminants in the northeast and northwest branches of the Anacostia River. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC. 88 pages.

Gruessner, B., D. Velinsky, J. Scudlark, T. Church, G. Foster and R. Mason. 1997. Dissolved and particulate transport of chemical contaminants in the Northeast and Northwest branches of the Anacostia River. Government of District of Columbia, Washington, DC. 64 pages.

Harshbarger, J.C., K.L. Price, E.B. May and A.E. Pinkney. 1997. Liver cancer in brown bullhead catfish from the Anacostia River, Washington, DC.

Haywood, H.C. and M. Focazio. 1990. Potomac River basin water quality 1986-1987. Interstate Commission on the Potomac River Basin, Rockville, MD. 85 pages.

Herson-Jones, L., A. Warner, B. Jordan and K. Hagan. 1994. Anacostia Watershed water quality report: 1987-1990. Metropolitan Washington Council of Governments, Washington, DC. 200 pages.

Hydro-Terra, I. 1989. Water quality and benthic macroinvertebrates in the lower Anacostia River, Washington, DC. Hydro-Terra Inc., Columbia, MD. 47 pages.

Hydro-Terra Inc. 1997. Data screening report #2, organic and inorganic analyses. Hydro-Terra, Inc., Columbia, MD.

Interstate Commission on the Potomac River Basin. 1994. In the Anacostia Watershed. Interstate Commission on the Potomac River Basin, Rockville, MD.

Interstate Commission on the Potomac River Basin. 1996. The Anacostia River toxics management action plan. Interstate Commission on the Potomac River Basin, Rockville, MD.

Kumble, P.A. 1990. The State of the Anacostia: 1989 Status Report. Metropolitan Washington Council of Governments, Washington, DC. 61 pages.

Loos, J. 1999. Contaminant concentrations observed in Anacostia River near the Benning Station after dredging. Summary of results.

LTI, L.-T., Inc. 1990. Sediment survey of priority pollutants in the District of Columbia waters. Interstate Commission on the Potomac River Basin, Rockville, MD. 12 pages + 2 appendices.

McCabe, W.M. 1997. An environmental characterization of the District of Columbia: A scientific foundation for setting an environmental agenda. U.S. Environmental Protection Agency - Region 3, Philadelphia, PA. 180 pages.

McDonald, N. 1998. Our unfair share II. Pollution in Washington, DC. African American Environmentalist Association, Washington, DC. 60 pages.

McDonald, N., D. Crain and J.D. Hair. 1994. Our unfair share. A survey of pollution sources in our nation's capital. African American Environmentalist Association, National Association of Neighborhoods, and National Wildlife Federation, Washington, DC. 106 pages.

Metropolitan Washington Council of Governments. 1986. Baseline water quality assessment of the Anacostia River. Metropolitan Washington Council of Governments, Department of Environmental Programs, Washington, DC. 101 pages.

Metropolitan Washington Council of Governments. 1991. A commitment to restore our home river. A six-point action plan to restore the Anacostia River. Metropolitan Washington Council of Governments, Department of Environmental Programs, Washington, DC.

Metropolitan Washington Council of Governments. 1995a. Anacostia special study/information packet. Metropolitan Washington Council of Governments, Washington, DC. 21 pages.

Metropolitan Washington Council of Governments. 1995b. Letter from Andy Warner, MWCOG, to Shane Ahn, EPA R3.

Metropolitan Washington Council of Governments and Naval Research Laboratory. 1998. Anacostia watershed toxics assessment. A collaborative, comprehensive approach.

Metropolitan Washington Council of Governments. 1998a. Anacostia watershed restoration progress and

conditions report 1990-1997. Anacostia Watershed Restoration Committee, Washington, DC. 42 pages.

Metropolitan Washington Council of Governments. 1998b. Unified Watershed Assessment and Watershed Priorities. Government of the District of Columbia, Water Quality Division, Environmental Health Administration, Bureau of Environmental Quality, Department of Health, Washington, DC. 37 pages.

Montaser, A. 1997. Impact assessment of landfill leachate on the Anacostia River water quality. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC. 15 + 6 appendices.

Murphy, D.R., A.E. Pinkney, R.E. Foley, P.C. McGowan, R. Li and L. Domico. 1998. Effects of wetland restoration using Anacostia River sediments at Kenilworth Marsh. U.S. Fish and Wildlife Service, Annapolis, MD. 200 pages.

PEPCO Potomac Electric Power Company. 1995. News Release: PEPCO joins D.C. Government in agreement to launch Anacostia River initiatives.

Phelps, H.L. No date. Biotoxicity of Anacostia River. Water and Sediments. Final Report to the District of Columbia Department of Environmental Services. 24 pages.

Phelps, H.L. 1985. Summer 1984 survey of mollusc populations of the Anacostia and Potomac rivers near Washington, DC. District of Columbia, Washington, DC. 50 pages.

Phelps, H.L. and K. Clark. 1988. Clam assay for toxic sediment at Kenilworth Marsh. Final technical report to Center for Urban Ecology, U.S. Park Service, Washington, DC. 21 pages.

Pinkney, G. 1999. Investigation of polynuclear aromatic hydrocarbon (PAH) and polychlorinated biphenyl (PCB) contamination at the Mason Neck National Wildlife Refuge complex: Linkages to tumors in Brown Bullhead and analysis of cytochrome P450 in Great Blue Heron.

Pinkney, A.E., W.H. Burton, L.C. Scott and J.B. Frithsen. 1993. An Assessment of potential residual effects of the January 1992 oil spill in the Anacostia River. Steuart Petroleum Company, Washington, DC. 300 pages.

Scatena, F.N. 1986. Recent patterns of sediment accumulation in the Anacostia River. Dept. of Geography and Environmental Engineering, Johns Hopkins University, Baltimore, MD.

Shepp, D.L. and H.H. Parikh. 1995. Hickey Run comprehensive pollution abatement program. Metropolitan Washington Council of Governments, Water Resource Management Division, Environmental Regulation Administration, Department of Consumer and Regulatory Affairs, Washington, DC. 12 pages + 3 appendices.

Shepp, D.L. and D.A. Cole. 1993. Hickey Run comprehensive pollution abatement study. Metropolitan Washington Council of Governments, Water Resource Management Division, Environmental Regulation Administration, Department of Consumer and Regulatory Affairs, Washington, DC. 8 pages + 2 appendices.

Stribling, J.B., E.W. Leppo and C. Daley. 1999. Biological Assessment of the Streams and Watersheds of Prince Georges County, Maryland. Prince Georges County, Maryland, Department of Environmental

Resources, Largo, MD.

U.S. Environmental Protection Agency Chesapeake Bay Program and U.S. Army Corps of Engineers. 1996. Biennial federal workplan for the Anacostia River watershed. U.S. Environmental Protection Agency Chesapeake Bay Program Office, Annapolis, MD. 63 pages + 3 appendices.

U.S. Environmental Protection Agency. 1997a. 1997 Environmental Justice Small Community Grants Project.

U.S. Environmental Protection Agency. 1998. Anacostia Ecosystem Initiative Update.

U.S. Environmental Protection Agency. 1999a. Progress under the Anacostia Ecosystem Initiative, Restoration for the River, Risk Reduction for the Community. U.S. Environmental Protection Agency, Region III, Philadelphia, PA. 4 pages.

U.S. Environmental Protection Agency. 1999b. The Anacostia Ecosystem Initiative. Chronology of Key Milestones. U.S. Environmental Protection Agency, Philadelphia, PA.

U.S. Environmental Protection Agency Chesapeake Bay Program. 1992. The Restoration of the Anacostia River. The Report to Congress. U.S. Environmental Protection Agency Chesapeake Bay Program Office, Annapolis, MD. .

U.S. Fish and Wildlife Service. 1994. Assessing sediment and pore water toxicity and biota response in the Anacostia River and Kingman Lake. U.S. Fish and Wildlife Service, Annapolis, MD. 20 pages.

U.S. Fish and Wildlife Service. 1997. Data report: Determination of toxicity and concentrations of inorganic and organic contaminants in sediments used to restore Kenilworth Marsh, Washington, DC. U.S. Fish and Wildlife Service, Branch of Water Quality and Environmental Contaminants, Annapolis, MD. 100 pages.

Velinsky, D.J., C. Haywood, T.L. Wade and E. Reinharz. 1992. Sediment contamination studies of the Potomac and Anacostia Rivers around the District of Columbia. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Hygiene Branch, Washington, DC. 147 pages.

Velinsky, D.J., J. Cornwell and G. Foster. 1994. Effects of dredging on the water quality of the Anacostia River. Interstate Commission on the Potomac River Basin, Rockville, MD. 70 pages.

Velinsky, D.J., T.L. Wade, C.E. Schlekat, B.L. McGee and B.J. Presley. 1994. Tidal river sediments in the Washington, D.C. area. I. Distribution and sources of trace metals. Estuaries. 17: 305-320.

Velinsky, D.J. and J.D. Cummins. 1994. Distribution of chemical contaminants in wild fish species in Washington, DC. Interstate Commission on the Potomac River Basin, Rockville, MD. 70 pages.
Velinsky, D.J. and J.D. Cummins. 1996. Distribution of chemicals in 1993-95 wild fish species in the District of Columbia. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC.

Velinsky, D.J., G.F. Riedel and G. Foster. 1999. Effects of stormwater runoff on the water quality of the tidal Anacostia River. U.S. EPA, Region III, Philadelphia. 132 pages.

Wade, T.L., D.J. Velinsky, E. Reinharz and C.E. Schlekat. 1994. Tidal river sediments in the Washington, D.C. area. II. Distribution and sources of organic contaminants. Estuaries. 17: 321-333.

Warner, A., D. Shepp, K. Corish and J. Galli. 1997. An existing source assessment of pollutants to the Anacostia watershed. The District of Columbia, Department of Consumer & Regulatory Affairs, Environmental Regulation Administration, Water Resources, Management Division, Washington, DC. 200 pages.

3. HYDRODYNAMIC PROCESSES/CHARACTERISTICS OF THE RIVER AND WATERSHED

Understanding hydrodynamic processes and characteristics of the Anacostia River and its watershed is the one of the first steps in identifying contaminant fate and transport information and general water quality. Hydrodynamics and water quality of a river are influenced by the characteristics of the river itself, as well as characteristics of the surrounding watershed. Processes include the effects of tides and the incursion of water from the Potomac River, river flow and velocity, inflows from tributaries, CSOs, NYPDES outfalls, and others, and storm responses (Section 3.1). General water quality characteristics and dynamics of the river can indicate impacts of land uses in the watershed (Section 3.2). In addition, water quality characteristics such as pH and dissolved oxygen can participate in metal cycling and the transport of organic compounds in aquatic systems (Allen, 1995 ; Schwarzenbach et al., 1993 ; Stumm and Morgan, 1981). An understanding of the watershed can be used to identify sources of loading observed in tributaries. In each of the subsections below, the data are reviewed in the context of a conceptual model of the river.

3.1 HYDRODYNAMIC CHARACTERISTICS OF THE RIVER

The tidal reach of the Anacostia River (the tidal Anacostia River) is bounded by the Northeast and Northwest Branches upstream, and the confluence with the Potomac River, 8.4 miles downstream (see Figure 3-1). Hydrodynamic characteristics of the river are an important component of a constituent transport model. Development of a hydrodynamic submodel requires understanding flow sources in the watershed. Investigations have been performed to support development of a hydrodynamic model, as discussed throughout this section. Flow, TSS and constituent data provide information for evaluation of mass transport.

The Anacostia River is freshwater with an average diurnal tidal amplitude of 3 feet (Warner et al., 1997). Tidal transport of sediment, including particulate bound contaminants in the Anacostia River would be expected to have cyclical directional fluctuations corresponding to the tide movement. On a broad scale, tides affect sediment dispersion in rivers. However, on a short time scale, tides can also affect advective transport of constituents[1] (Chapra, 1997). Deposition would be expected to occur under high, slack tide periods when water velocities are minimal and water column particulate loading is maximum. Slack tide periods occur during the period between flow and ebb tides. Resuspension or scouring may occur during ebb tides when water movement out from the river occurs due to the outward flow of the tide combined with the flow of the river. The tidal influence complicates interpretation of sediment movement and depositional patterns.

The tidal Anacostia River has been characterized as analogous to a tidal lake that occasionally receives enough discharge to respond like a river (Scatena, 1986). It has been estimated that approximately 85% of the sediment that enters remains in the tidal river (Scatena, 1986). The average residence time of water in

[1] Constituents refer to chemicals that occur in the environment originating from natural or anthropogenic sources.

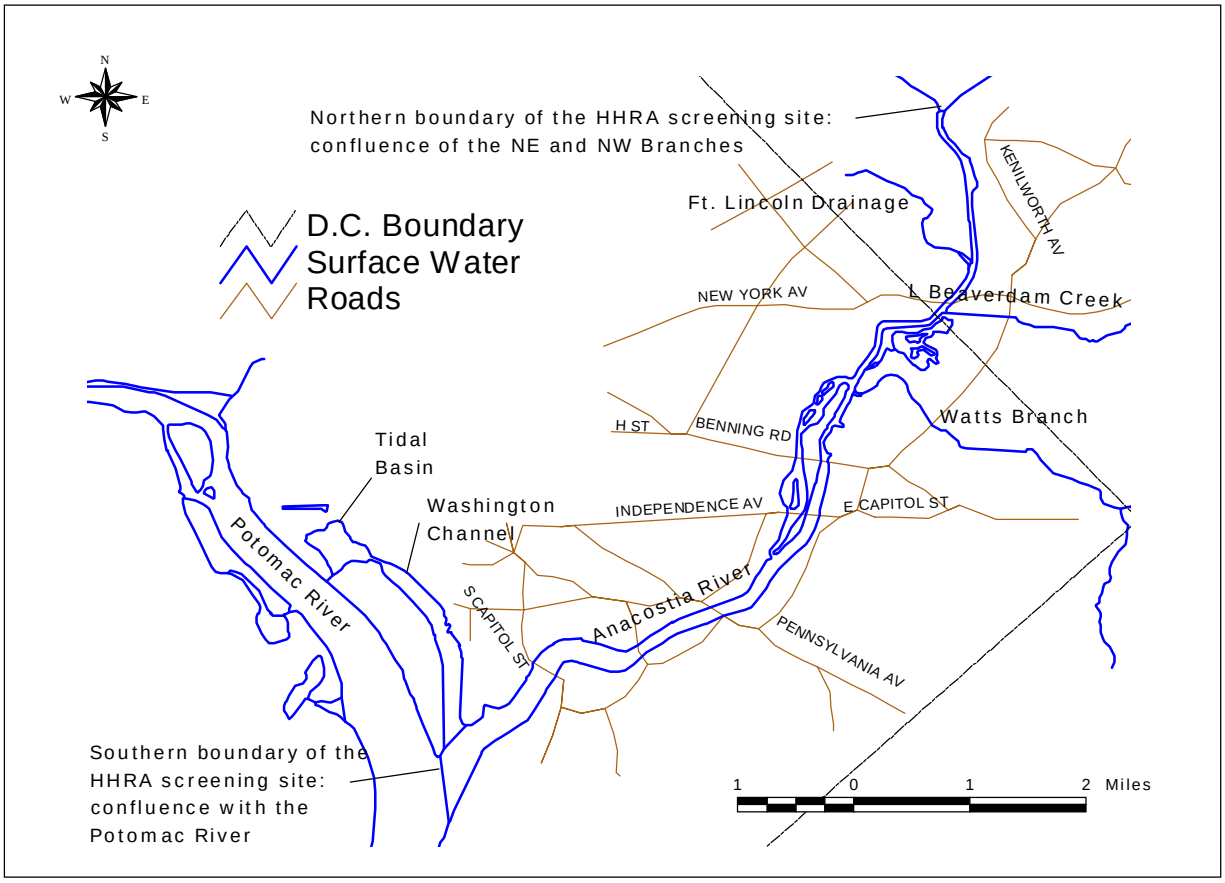


Figure 3-1. Site Map for the Tidal Anacostia River and the Human Health Risk Screening Assessment. For the purpose of the human health screening assessment, the site was geographically defined as the tidal Anacostia River extending from the juncture of the Northwest and Northeast Branches to the Potomac River, including Kenilworth Marsh and Kingman Lake, and excluding the Washington Channel and Tidal Basin

the lower Anacostia River, south of Blandensburg, is approximately 35 days (Nemura and Pontikakis-Coyne, 1991, cited in Gruessner et al., 1997). The generally slow movement of water in the river is an important hydrodynamic characteristic in the transport of solids and potential contaminants.

Current velocities and flow are hydrologic properties used to assess mass loading and transport of solids or contaminants in a river. Current velocity (distance/time) is an important physical property that affects the potential for particulate matter settling, resuspension, or scouring. The product of current velocity and the cross-sectional area of the river at a location are used to calculate flow (volume/time). If concurrently collected flow and constituent concentration data are available, mass transport estimates (mass/time) may be calculated as their product. Mass loading may be calculated as the difference in mass transport between two sections of a river. In a river, current velocities, flow, and constituent loading and transport are dynamic. Therefore, changes in current velocity result in changes in transport potential. Constituent mass loading in a river may be highly variable. Therefore, the accuracy of mass transport estimates is dependent on the representativeness of sampling data.

In the Anacostia River, the average current velocity decreases in the region of the river from Pennsylvania Avenue Bridge to the vicinity of the 11th Street Bridge (Table 3-1). The decrease in current velocity has been proposed as a potential mechanism for increased settling of suspended matter in this area, relative to upstream higher velocity locations (Coffin et al. 1999).

Table 3-1. Average River Current Velocities for Select Sections of the tidal Anacostia River

Sampling location	Avg Velocity (ft/s)	Number of Measurements
Benning Road Bridge	0.72 +/- 0.66	89
Pennsylvania Avenue Bridge	0.43 +/- 0.26	71
The vicinity of the 11th Street Bridge	0.26 +/- 0.16	113
Confluence with Potomac River	0.69 +/- 0.62	115

Note: The current velocity data were collected at 0.5m depth intervals at each station throughout the water column.

Source: Coffin et al., 1999

At the Benning Road Bridge (upstream) and the confluence with the Potomac River (downstream) sampling stations, higher variability in current velocities were observed relative to the other stations. The differences were attributed to tidal effects on a narrow channel, and confluence with the Potomac River, respectively (Coffin et al., 1999). During a slack tide, current velocity rates measured at mid-river stations ranged from 0 to 0.3 ft/sec. As noted above, increased settling may occur during slack tides.

The main flow (the thalweg) occurs in the deepest portions of a river (Rutherford, 1994). Traveling from

upstream to downstream, the main flow of the Anacostia River is located in the approximate center of the river at Benning Road Bridge and meanders toward the west portion of the channel in the vicinity of the Pennsylvania Avenue Bridge. Further downstream, in the vicinity of the 11th Street Bridge, the main flow meanders toward the southeast shore and then back toward the center of the channel at the confluence of the Anacostia River with the Potomac River (Coffin et al., 1998). Areas outside the main flow tend to have lower current velocities facilitating settling of suspended solids.

Approximate mean annual flows for the tidal Anacostia River are summarized in Table 3-2 .

Table 3-2. Approximate Mean Annual Flows for the Anacostia River

Year	Flow (cfs)	Range	Data source
Oct 90-Sep 92	120	50 (Aug 91) - 260 (Mar 91)	DC 1994 [1]
1993	180	50 - 530 cfs (1993–1997)	Kelly 1998 [1]
1994	175		Kelly 1998
1995	125		Kelly 1998
1996	250		Kelly 1998
1997	140		Kelly 1998

[1] Anacostia River flows obtained as the sum of flows at the Northeast and Northwest Branches as measured by the United States Geological Survey (USGS). Flow values were adjusted by a factor of 1.02 to account for ungaged drainage area (DC, 1994; Kelly, 1998).

Cfs, cubic foot per second

Flow data from bordering water bodies also provides information on the hydrodynamics of the Anacostia River. Upstream of the tidal Anacostia River, at United States Geological Survey (USGS) gages located in the Northeast and Northwest Branches, long-term mean daily flows indicated that the tributaries contributed approximately 64% (85 cfs) and 36% (48 cfs) of the total flow to the tidal Anacostia River at Bladensburg, respectively (James et al., 1995 cited in Gruessner et al., 1997). Estimates of flow from the upper portions of the river upstream of the gaging stations include watershed contributions, tributaries, CSOs, NPDES outfalls and other sources within those areas.

Downstream, annual mean flow rates in the Potomac River at Chain Bridge for 1991 and 1992 were 1,200 cfs and 800 cfs, respectively (DC, 1994). Flow variation in the Potomac River may influence tidal affects in the Anacostia River, as discussed above. Over that time period, flow ranged from approximately 100 cfs (Sep, Oct 91) to 3,500 cfs (Jan 91) (DC, 1994). Information on flow dynamics of Kingman Lake and the Kenilworth Marsh located in the tidal reach of the Anacostia River were not identified in the documents reviewed. However, it was noted that both the lake and the marsh were almost entirely constructed (Scatena, 1986). Current velocities would be expected to be lower in the lake and in the marsh compared to the main stem of the river, which may result in a higher deposition and burial rates (mass/time). However, the flow characteristics of the lake and marsh need to be understood to evaluate this possibility.

Storm and flood events result in periods of sediment loading, resuspension, and deposition in river

systems (Huber, 1993 ; Rutherford, 1994). The initial pulse of water that occurs during a storm event will often carry accumulated materials from the watershed into the river at higher concentrations than found during other periods of the storm event (Huber, 1993). This first flush phenomenon can make an important contribution to the transport of sediments and sediment-laden contaminants. Therefore, storm and flood event characterization should include evaluation of constituent concentrations as the river flow rises during a storm (the rising limb of a hydrograph). As river flows peak, flood plains may be impacted by inundation with water. As the flows subside, particulate matter may be deposited as flow velocities decrease, allowing settling to occur.

Limited storm sampling has been performed in the Anacostia River system. Storm samples that have been collected consisted of sampling before and after the storm events (Gruessner et al., 1997; Velinsky et al., 1999). Therefore, first flush of the storms was not sampled. As a result, available storm sampling data for the Anacostia River provide a qualitative rather than quantitative assessment of the impacts of storm events on water quality.

A notable storm event occurred in the Washington, DC area on May 6, 1989. The storm event produced the highest flows recorded since 1979. As a result of the storm, daily discharges from the tidal Anacostia River of approximately 5,800 cfs were reported (MWCG-DEP, 1990). The elevated flows that occurred during the May 1989 storm likely resulted in elevated sediment transport at that time (Huber, 1993 ; Rutherford, 1994).

In addition to rain storm events, snowfall events and subsequent snow melt may also elevate flows and increase sediment transport in the river. In the Anacostia River watershed snowmelt may not be a concern. Although, in northern climates, this type of event can be a significant factor in the hydrodynamics of a river. Information was not identified that would confirm or allay concerns of the potential impact of snow events on the Anacostia River.

3.2 GENERAL WATER QUALITY CHARACTERISTICS

General water quality of a river is assessed by several parameters. Total suspended solids (TSS) is one of the most important parameters that is used along with flow data to measure solids loading to the river (Section 3.2.1). Solids are often a carrier for contaminant transport of hydrophobic organic compounds and metals that associate with or are adsorbed to particulate matter. Dissolved organic carbon (DOC) and particulate organic carbon (POC) are useful for evaluating adsorption potential of the solids, as well as the aquatic productivity of the river (Section 3.2.2). Conventional water quality parameters consisting of pH, conductivity, dissolved oxygen, and turbidity are also used to evaluate the water quality of the river (Section 3.2.3). Water temperature profiles can be useful to refine estimates of transport (Section 3.2.4). Nutrient data provide a characterization of general water quality (Section 3.2.5). Organic compound and metals data are also summarized (Section 3.2.6).

3.2.1 TOTAL SUSPENDED SOLIDS

Total suspended solids (TSS) in the water column are a key component of evaluations of sediment and constituent transport in rivers (Manhattan College, 1994). Transport and deposition of solids is dynamically related to flow and watershed characteristics. Results of TSS sampling indicated that average concentrations ranged from 19 to 39 mg/L along the length of the tidal Anacostia River (Table 3-3). Typical ranges for TSS in rivers in streams are 10–110 mg/L (McCutcheon et al., 1993). The highest TSS concentrations were consistently measured in upper reaches of the tidal Anacostia River from the Pennsylvania Avenue Bridge to the Blandensburg Marina. Lower concentrations were observed in the vicinity of the 11th Street Bridge (Coffin et al., 1999). The relative spatial distribution of TSS is consistent with expected scouring and loading from upstream sections of the river with higher current velocities, and settling and deposition in lower reaches that also have lower current velocities (Table 3-1). Seasonal variations in TSS concentrations have also been observed in the Anacostia River ranging from 2.1 to 69.0 mg/L (Table 3-3) (Coffin et al., 1999).

Table 3-3. Average Total Suspended Solids Concentration

Month/Year	Concentration (mg/L)	Number of Observations
June 1997	19.4 +/- 6.8	38
September 1997	20.0 +/- 12.2	49
February 1998	38.8 +/- 5.6	12
May 1998	25.0 +/- 9.6	20

Source: Coffin et al., 1999

Non-point sources of TSS consist of in-stream erosion (bank erosion and substrate scour), runoff of solids deposited on paved areas, and construction activities which account for 95% of the TSS loading to the Anacostia and its tributaries (Warner et al., 1997). TSS loading in the Anacostia River is estimated at 48,200 tons (96 million pounds) per year, averaging approximately 0.43 tons per acre per year (Warner et. al., 1997). Generally, the largest subwatersheds contributed the largest TSS loading and most developed areas contributed the highest average TSS loadings. During storm events it was estimated that approximately 95% of the TSS in the Anacostia watershed resulted from stormwater runoff, the remaining 5% was associated with Combined Sewer Overflow (CSO) discharges (Warner et al., 1997)

General spatial trends of water column concentrations of TSS in the river indicated that TSS increased in the vicinity of Kenilworth Marsh and the CSX railroad bridge and then declined downstream (Velinsky et al., 1999). In addition, occasional increases in TSS concentration were observed at the mouth of the Anacostia River. Following storm events, increases in water column TSS concentrations generally occurred in the river (Velinsky et al., 1999). The effects of tides on the dynamics of TSS concentrations in the Anacostia River was not evaluated in the reports reviewed. Water column data did not include

references to tide levels observed during sampling.

3.2.2 DISSOLVED ORGANIC CARBON AND PARTICULATE ORGANIC CARBON

Organic carbon in aquatic environments may play an important role in the transport of organic compounds in the water column. Many organic compounds are nonpolar and do not readily interact with water. However, naturally occurring organic carbon from plants and organisms in the water column may provide a more favorable interactive media for the transport of organic compounds (Schwarzenbach et al., 1993)

Similar to observations of spatial trends of TSS (Section 3.2.1), water column concentrations of POC increased in the vicinity of Kenilworth Marsh and the Conrail railroad bridge and declined downstream. Generally, increases occurred in water column POC concentrations following storm events at downstream locations, although this trend was not observed consistently at stations located upstream of the Anacostia Bridges (Station 3; Velinsky et al. 1999).

3.2.3 CONVENTIONAL WATER QUALITY PARAMETERS

Water quality characteristics such as pH and dissolved oxygen can participate in metal cycling and the transport of organic compounds in aquatic systems (Allen, 1995 ; Schwarzenbach et al., 1993 ; Stumm and Morgan, 1981).

3.2.4 THERMAL STRATIFICATION

Thermal stratification may occur in water bodies due to the density difference of waters with different temperatures. Thermal stratification is common in water bodies with slow water movement, particularly in lakes. It may also occur in slow moving rivers. Stratification decreases circulation of water depths in a river or lake thereby impacting water movement and fine particulate transport. The overall effect of thermal stratification is to reduce the rate of vertical mixing and in some cases this reduction can be substantial (Rutherford, 1994).

Thermal stratification was observed in the Anacostia River at sampling stations at the Pennsylvania Avenue Bridge and in the vicinity of the 11th Street Bridge. The maximum gradient was located in the vicinity of the 11th Street Bridge with a change of 1.3 degrees Celsius that was observed through a 2 m depth (Coffin et al., 1998). During an ebb tide, the thermal stratification was less pronounced. Seasonal differences indicated that stratification decreased in winter months (Coffin et al., 1999).

The thermal stratification data collected to date has qualitatively identified stratification in the lower reach of the tidal Anacostia River during certain time periods. The effect of thermal stratification on constituent transport has not been completely identified. However, the presence of thermal stratification is indicative of the tidal influence of the Potomac River in lower reaches of the river.

3.2.5 WATER COLUMN PARTICULATE MATTER

Particle size analysis

Particle size analysis provides a description of the materials contained in the water column thereby providing evidence of flow dynamics. Data collected in June 1997 indicated that the amounts of small particle size (0.2 to 1.0 :m) materials in the water column in the vicinity of Benning Road Bridge and the vicinity of the 11th Street Bridge were approximately 60% lower than in areas at the mouth of the river. This suggests that settling of fine materials occurs in this region of the river. For all sections sampled in the river, the predominant particle size was in the range of 3.0 to 5.0 :m (Coffin et al. 1998). The particle size distribution analysis was performed by acoustic signal analysis. Horizontal layer scattering was observed at all sites, supporting the observations of stratification due to thermal and density differences that occur in the water column (Coffin et al. 1998).

Seston

Seston consists of minute living organisms and particles of nonliving organic matter which float in the water and contribute to turbidity. Therefore, seston analyses provide a measurement of the organic carbon content of river sediment. Concentrations of seston measured across the river were skewed toward the west shore at stations located at the Benning Road and Sousa Bridges. In the vicinity of the vicinity of the 11th Street Bridge, concentrations were consistent across the river. Further downstream, at the confluence with the Potomac River, concentrations were skewed toward the east shore (Coffin et al. 1998). Overall, spatially traveling down the river, seston concentrations decreased (Coffin et al. 1998). The decreases in seston concentrations observed were likely related to decreases in current velocities that resulted in increased settling of suspended materials as the water traveled downstream from a region of relatively high current velocity to a region of lower current velocity. However, TSS monitoring was not included in the study to evaluate this possible relationship.

3.3 DATA GAPS RELATED TO MODELING RIVER HYDRODYNAMICS

The hydrodynamics in the river are not understood sufficiently to enable predictions of contaminant concentration profiles over the length of the river, or to identify future high impact areas and estimate associated concentrations. This limitation is particularly relevant to extreme (high and low) flow conditions and disturbances that might be imposed on the river as part of remediation or other modifications to the river (e.g., dredging). Some specific data gaps include the following:

X Water flows and channel volumes have not been determined over a sufficient geographic and temporal scale to support the development of a hydrodynamic model of both the "average" long-term behavior of the river and the behavior of the river during extreme events (e.g., storms, drought).

X Flows and first-flush and peak fluvial chemical loadings from tributaries to the tidal Anacostia during storm events have not been quantified in available documents.

X The exchange of surface water and sediment between the Potomac and Anacostia Rivers during tidal flux has not been quantified.

X The exchange of ground water and surface water within the tidal Anacostia River has not been quantified.

X A model calibration data set has not been collected. This would include sediment and water column concentrations of representative chemicals at various locations in the river, including predicted high impact areas, at various times, including during and after extreme events (e.g. storms).

REFERENCES FOR CHAPTER 3

Allen, H., ed.. 1995. Metal Contaminated Aquatic Sediments. Ann Arbor Press, Inc. Chelsea, Michigan..

Badruzzaman, A.B.M. and A.D. Nemura. 1993. Kingman Lake Water Quality Model. Metropolitan Washington Council of Governments, Environmental Regulation Administration, Department of Consumer and Regulatory Affairs, Washington, DC. 39+ 4 appendices.

Brush, G.S., E.A. Martin, R.S. DeFries, C.A. Rice. 1982. Comparison of Pb210 and Pollen methods for determining Rates of Estuarine Sediment Accumulation. Quaternary Research 8:2, 192-197.

Chapra, S. C. 1997. Surface Water Quality Monitoring. The McGraw-Hill Companies, Inc. New York.

Coffin, R., J. Pohlman, and C. Mitchell. 1999. Fate and Transportation of PAH and Metal Contaminants in the Anacostia River Tidal Region. Navel Research Laboratory, NRL/MR/6110C99-8327.

Coffin, R., M. Orr, E. Cary, L. Cifuenties, and J. Pohlman. 1998. Contaminant Distribution and Fate in Anacostia River Sediments: Particulate Transport Study. Navel Research Laboratory, NRL/MR/6115C98-8139.

Government of the District of Columbia Department of Health, Environmental Health Administration. 1998. The Navy's use of PCBs and contribution to the PCB problem in the District's Rivers.

Government of the District of Columbia Department of Health, Environmental Health Administration. 1998. 1998 Water Quality Report to U.S. EPA and Congress.

Government of the District of Columbia, Department of Consumer & Regulatory Affairs. 1997. Determination of the volume of contaminated sediments in the Anacostia River. Authors: D. Velinsky, B. Gruessner, C. Haywood, J. Cornwell, R. Gammish, and T. Wade.

Government of the District of Columbia Department of Health, Department of Consumer & Regulatory Affairs. 1994. The District of Columbia Water Quality Assessment. 1994 Report to the Environmental Protection Agency and U.S. Congress.

Gruessner, B., D. Velinsky, G. Foster, J. Scudlark, and R. Mason. 1997. Dissolved and Particulate Transport of Chemical Contaminants in the Northeast and Northwest Branches of the Anacostia River. Report submitted to Department of Consumer and Regulatory Affairs.

Huber, W. C. 1993. Contaminant Transport in Surface Water. In Handbook of Hydrology, David R. Maidment, ed. McGraw-Hill, Inc. New York.

Kelly, M.N. and T.J. Gordon. 1998. 1998 Water Quality Report to U.S. EPA and Congress. Government of the District of Columbia Department of Health, Environmental Health Administration.

Maidment, D. R.(ed.). 1993. Handbook of Hydrology. McGraw-Hill, Inc. New York.

Manhattan College. 1994. Modeling of Transport, Fate, and Bioaccumulation of Toxic Substances in Surface Water. Riverdale, New York. Thirty-Ninth Institute in Water Pollution Control.

McCutcheon, S., J. Martin, and T. Barnwell. 1993. Water Quality. In Handbook of Hydrology, David

Maidment, ed. McGraw-Hill. New York.

Metropolitan Washington Council of Governments. 1990. The State of the Anacostia.

Rutherford, J. C. 1994. River Mixing. John Wiley and Sons Ltd. New York.

Scatena, F. 1986. Recent Patterns of Sediment Accumulation in the Anacostia River. Johns Hopkins University. Draft Report submitted to Water Hygiene Branch of the D.C. Environmental Control Division.

Schwarzenbach, R., P. Gshwend, and D. Imboden. 1993. Environmental Organic Chemistry. John Wiley & Sons, Inc. New York.

Stumm, W. and J. Morgan. 1981. Aquatic Chemistry: An Introduction to Chemical Equilibria in Natural Waters. 2nd Edition. John Wiley & Sons. New York.

United States Environmental Protection Agency. 1997. A Scientific Foundation for Setting an Environmental Agenda, An Environmental Characterization of the District of Columbia. U.S. EPA Region 3. April 30, 1997.

Velinsky, D., C. Haywood, T. Wade, and E. Reiharz. 1992. Sediment Contamination Studies of the Potomac and Anacostia Rivers around the District of Columbia. Report submitted to DCRA, Water Hygiene Branch. ICPRB Report No. 92-2.

Velinsky, D., B. Gruessner, C. Haywood, J. Cornwall, R. Gammish, and T. Wade. 1997. Determination of the volume of Contaminated Sediments in the Anacostia River; District of Columbia. Report submitted to the Department of Consumer and Regulatory Affairs. Grant No. 94g-95-WRMD01.

Velinsky, D., G. Reidel, and G. Foster. 1999. Effects of Storm Water Runoff on the Water Quality of the tidal Anacostia River. Report submitted to U.S. EPA - Region III, Water Protection Division. March 1, 1999.

Warner, A., D. Schepp, K. Corish, and J. Galli. 1997. An Existing Source Assessment of Pollutants to the Anacostia Watershed. Report prepared for The District of Columbia, Department of Consumer & Regulatory Affairs. June 1997.

4. SEDIMENT TRANSPORT DYNAMICS

Sediment transport dynamics of the tidal Anacostia River are described herein. Key issues in developing a sediment transport model are bed-mapping, *in situ* resuspension potential, and bed elevation monitoring (Manhattan College, 1994). Water column TSS concentrations which were discussed previously (Section 3.2.1), are an additional key component to the sediment transport model (Manhattan College, 1994). Sediment bed-mapping and bed elevation monitoring provide a description of depositional areas in the river and the accumulation of sediment over time (Section 4.1). The dynamics of sediment transport has been impacted by dredging in the river (Section 4.2). An understanding of the physical characteristics of the sediment in the river provides a basis for evaluating *in situ* resuspension potential (Section 4.3). A conceptual model describing the pathways of constituent influx and migration through the tidal Anacostia River has been developed incorporating existing data for the river (Section 6). For each of the subsections below, data are reviewed in the context of this conceptual model.

4.1 DEPOSITIONAL AREAS

Sediment deposition occurs in low velocity regions of the river. As discussed previously, deposition is dynamic due to fluctuations in flow (Section 2.1).

The volume of contaminated sediment and burial rates of the sediment were investigated in the Anacostia River. Sediment depths of up to 3 meters extending across most of the lower tidal Anacostia River were reported. Several studies have been conducted to evaluate sediment depositional rates:

X Analysis of burial rates using lead-210 dating techniques indicated a sediment accumulation rate in the Washington Channel of approximately 0.9 cm/yr for the period of 1878–1978 (Brush et al. 1982, cited in Velinsky 1997).

X Recent sedimentation rates varied between 0.9 to 1.6 cm/yr. From 1972–1985, the sedimentation rate for the Anacostia River was estimated as approximately 3.1 cm/yr (Velinsky et al., 1997).

X The rate of sediment deposition in the mid-channel was estimated as 4.2 cm/yr for the period 1958 to 1980 (Scatena, 1986). The estimate was based on 12 cross-section surveys conducted at a location in the tidal Anacostia River. That estimate agreed with an estimate based on the mass balance of TSS in the tidal embayment. The total amount of sediment deposited evenly over the entire tidal area was estimated to be 3.2 $g/cm^2/yr$ (Scatena, 1986). This was estimated to be equivalent to a sedimentation rate of 3.8 cm/yr (wet) and 1.9 cm/yr (dry) (Scatena, 1986). A range of sedimentation rates from 1.4 to 8.0 cm/yr was estimated to account for annual variabilities (Scatena, 1986).

X Sediment (measured as seston in sediment traps) deposition rates were also estimated for June 26, 1997. From that study, the highest sedimentation rates were observed in the upper river at Benning Road Bridge, 1150 mg/d. Lower values were observed in the vicinity of the 11th Street Bridge with an average of 170 mg/d. A rapid change in sedimentation was observed over the short distance between the Pennsylvania Avenue Bridge and the vicinity of the 11th Street Bridge. The observed differences were attributed to tidal mixing, current flow rates and the effect of river morphology

(Coffin et al., 1998). The tide status was not reported in the report.

In summary, most estimates of sediment deposition rates in the Anacostia River were between 0.5 and 4.2 cm/yr. Differences in reported sedimentation rates may be due to techniques employed, or spatial or annual differences.

Sediment mass balances indicated that approximately 85% of sediment that enters the lower river is trapped and buried (Scatena, 1986). However, using lower estimates of sediment deposition rates obtained by others (Coffin et al., 1998 ; Velinsky et al., 1997) could reduce this retention rate to less than 50%. It was also estimated that 30–40% of the total annual amount of sediment deposited occurred during distinct hydrologic events rather than continuous sedimentation (Scatena, 1986).

High resolution seismic-reflection profiling was conducted to evaluate bathymetry of the river bed. Eight sediment cores were also collected (Velinsky et al., 1997). The high resolution profiles were not available for review.

Grain size analysis is used for estimating deposition. If grain size analysis is used to estimate the concentrations of chemical constituents in the Tidal Anacostia River, it will not provide information that is sufficient to identify separate sources of the chemical constituents. Some of the confounders that preclude the use of grain size analysis to identify specific sources include variability in the intensity of chemical releases over time and variability in the initial time and duration over which releases occur. In addition, NAPL sources will not be successfully characterized using grain size analysis alone.

4.2 DREDGING

The Anacostia River has been dredged for various purposes. Channel improvement projects in the Anacostia River started in the 1880s with the removal of polluted sediments from the Anacostia River and Washington Channel area. In the late 1950s, the river was structurally modified to its present configuration upstream to Blandensburg, Maryland (Scatena, 1986). Past dredging of the center of the river resulted in depths outside of the channel generally ranging from 0.5 to 5 m (Velinsky et al., 1992). River dredging alters the hydrodynamics of the river, generally increasing flow in the center of the channel and reducing flow outside of the channel. Changes in sediment transport would likely result from the hydrologic impacts.

More recently, dredging has been performed at Blandensburg Marina. A study conducted to evaluate the potential impacts of dredging at the Blandensburg Marina concluded that dredging of the marina did not impact surface water quality of the upper tidal Anacostia River during dredging (Velinsky et al., 1994 a).

4.3 SEDIMENT CHARACTERISTICS

Sediment has two primary origins. It may originate from the drainage basin (allochthonous) or from photosynthetic processes (autochthonous). These sediment types can often be distinguished by organic carbon content, density, and particle size. The autochthonous solids are generally higher in organic carbon content and more reactive than allocthonous sediment. They are less dense with high water content and tend to be smaller in size (Chapra, 1997). Therefore, the two types of sediment also tend to have different transport dynamics. Sediment characterization of depositional areas requires investigation of river bed surface sediment and subsurface sediment to obtain a profile of materials that have been deposited over time.

Grain size distribution

Grain size distribution analyses of sediment in the river identified predominantly clay and silt (<63 :m). In contrast, sewer samples had a greater range in grain size than those collected in the river. The size differences were attributed to physical sorting of particles in the sewers due to relatively high current velocities associated with storm runoff in sewers (Velinsky et al., 1992).

Total Organic Carbon

Analysis of total organic carbon (TOC) content of sediment supports interpretation of sediment source and affinity of the sediment for binding chemicals, as well as the aquatic productivity of the river. In the Anacostia River, TOC concentrations range from 2.5 to 6.4% of the sediment on a dry weight basis and it averages 4.0 +/- 0.9%. The highest concentrations of TOC measured in the river sediment were at Kingman Lake and in the tidal basin (Velinsky et al., 1994b). These areas represent areas of greater aquatic productivity, compared to other portions of the tidal Anacostia river. They are also areas that would be expected to have relatively lower current velocities and higher sediment deposition rates compared to other sections of the river.

Outfalls along the Anacostia River contained concentrations of TOC ranging from 0.7% to 11% indicating a wide range of point source contributions of TOC and physical sorting of particles (Velinsky et al., 1994b). In contrast, TOC concentrations in sewers were lower, averaging approximately 1.0±0.9% TOC. The lower concentrations of TOC in sewers were attributed to the larger grain size of the sand encountered compared to outfall samples (Velinsky et al., 1994 b). The distinction between outfall and sewer samples was not clear from review of the report.

4.4 DATA GAPS RELATED TO MODELING SEDIMENT TRANSPORT

The dynamics of sediment transport in the river are not understood sufficiently to enable predictions of contaminant concentration profiles over the length of the river, or to identify future high impact areas and estimate associated concentrations. This limitation is particularly relevant to extreme (high and low) flow conditions and disturbances that might be imposed on the river as part of remediation or other modifications to the river (e.g., dredging). Some specific data gaps include the following:

X Water flows and channel volumes have not been determined over a sufficient geographic and temporal scale to support the development of a hydrodynamic model of both the "average" long-term behavior of the river and the behavior of the river during extreme events (e.g., storms, drought).

X The exchange of surface water and sediment between the Potomac and Anacostia Rivers during tidal flux has not been quantified.

X Depositional patterns of sediments transported into the tidal Anacostia from tributaries, including the Northeast and Northwest branches, during and between storm events have not been characterized.

X Conditions under which deposited sediments are resuspended and transported, and the relative importance of this mechanism for transport of chemical contaminants, have not been characterized.

X Particulate deposition on the floodplain and tributaries, particularly within Kennilworth Marsh and Kingman Lake, during large flow events such as storm events or during spring snowmelt have not yet been channelized has not been addressed relative to human exposures to chemical contaminants.

X The frequency and extent of dredging deep tidal river sediments have not been characterized in available documents; deep dredging may promote the resuspension of formerly buried contaminated sediment.

X Partitioning of COPCs between sediment and surface water needs to be better charactorized.

X A model calibration data set has not been collected. This would include sediment and water column concentrations of representative chemicals at various locations in the river, including predicted high impact areas, at various times, including during and after extreme events (e.g. storms).

REFERENCES FOR CHAPTER 4

Allen, H., ed.. 1995. Metal Contaminated Aquatic Sediments. Ann Arbor Press, Inc. Chelsea, Michigan..

Chapra, S. C. 1997. Surface Water Quality Monitoring. The McGraw-Hill Companies, Inc. New York.

Coffin, R., J. Pohlman, and C. Mitchell. 1999. Fate and Transportation of PAH and Metal Contaminants in the Anacostia River Tidal Region. Navel Research Laboratory, NRL/MR/6110C99-8327.

Coffin, R., M. Orr, E. Cary, L. Cifuenties, and J. Pohlman. 1998. Contaminant Distribution and Fate in Anacostia River Sediments: Particulate Transport Study. Navel Research Laboratory, NRL/MR/6115C98-8139.

Government of the District of Columbia Department of Health, Environmental Health Administration. 1998. The Navy's use of PCBs and contribution to the PCB problem in the District's Rivers.

Government of the District of Columbia Department of Health, Environmental Health Administration. 1998. 1998 Water Quality Report to U.S. EPA and Congress.

Government of the District of Columbia, Department of Consumer & Regulatory Affairs. 1997. Determination of the volume of contaminated sediments in the Anacostia River. Authors: D. Velinsky, B. Gruessner, C. Haywood, J. Cornwell, R. Gammish, and T. Wade.

Government of the District of Columbia Department of Health, Department of Consumer & Regulatory Affairs. 1994. The District of Columbia Water Quality Assessment. 1994 Report to the Environmental Protection Agency and U.S. Congress.

Gruessner, B., D. Velinsky, G. Foster, J. Scudlark, and R. Mason. 1997. Dissolved and Particulate Transport of Chemical Contaminants in the Northeast and Northwest Branches of the Anacostia River. Report submitted to Department of Consumer and Regulatory Affairs.

Huber, W. C. 1993. Contaminant Transport in Surface Water. In Handbook of Hydrology, David R. Maidment, ed. McGraw-Hill, Inc. New York.

Maidment, D. R.(ed.). 1993. Handbook of Hydrology. McGraw-Hill, Inc. New York.

Manhattan College. 1994. Modeling of Transport, Fate, and Bioaccumulation of Toxic Substances in Surface Water. Riverdale, New York. Thirty-Ninth Institute in Water Pollution Control.

McCutcheon, S., J. Martin, and T. Barnwell. 1993. Water Quality. In Handbook of Hydrology, David Maidment, ed. McGraw-Hill. New York.

Metropolitan Washington Council of Governments. 1990. The State of the Anacostia.

Rutherford, J. C. 1994. River Mixing. John Wiley and Sons Ltd. New York.

Scatena, F. 1986. Recent Patterns of Sediment Accumulation in the Anacostia River. Johns Hopkins University. Draft Report submitted to Water Hygiene Branch of the D.C. Environmental Control Division.

Schwarzenbach, R., P. Gshwend, and D. Imboden. 1993. Environmental Organic Chemistry. John Wiley

& Sons, Inc. New York.

Stumm, W. and J. Morgan. 1981. Aquatic Chemistry: An Introduction to Chemical Equilibria in Natural Waters. 2nd Edition. John Wiley & Sons. New York.

United States Environmental Protection Agency. 1997. A Scientific Foundation for Setting an Environmental Agenda, An Environmental Characterization of the District of Columbia. U.S. EPA Region 3. April 30, 1997.

Velinsky, D., G. Reidel, and G. Foster. 1999. Effects of Storm Water Runoff on the Water Quality of the tidal Anacostia River. Report submitted to U.S. EPA - Region III, Water Protection Division. March 1, 1999.

Velinsky, D., B. Gruessner, C. Haywood, J. Cornwall, R. Gammish, and T. Wade. 1997. Determination of the volume of Contaminated Sediments in the Anacostia River; District of Columbia. Report submitted to the Department of Consumer and Regulatory Affairs. Grant No. 94g-95-WRMD01.

Velinsky, D., J. Cornwell, and G. Foster. 1994a. Effects of Dredging on the Water Quality of the Anacostia River. Report submitted to Water Quality Control Branch, Environmental Regulation Division. Grant No. 92g-92-WRMD06.

Velinsky, D., T. Wade, C. Schlerkat, B. McGee, and B.J. Presley. 1994b. Tidal River Sediments in the Washington, D.C. Area. I. Distribution and Sources of Trace Metals. Estuaries V17:2, 305-320. June 1994.

Velinsky, D., C. Haywood, T. Wade, and E. Reiharz. 1992. Sediment Contamination Studies of the Potomac and Anacostia Rivers around the District of Columbia. Report submitted to DCRA, Water Hygiene Branch. ICPRB Report No. 92-2.

Warner, A., D. Schepp, K. Corish, and J. Galli. 1997. An Existing Source Assessment of Pollutants to the Anacostia Watershed. Report prepared for The District of Columbia, Department of Consumer & Regulatory Affairs. June 1997.

5. EXISTING DATA SUMMARY/COMPILATION

5.1 CONSTRUCTION OF DATABASE FOR HUMAN HEALTH RISK SCREENING ASSESSMENT

A database was created using Microsoft Access to facilitate the human health risk screening assessment (HHRA screening database). The database included information contained in the Anacostia River Watershed Database and Mapping Project (NOAA, 2000) and water quality data that was provided by NOAA in spreadsheet format on January 3, 2000; the latter is from a study by Velinsky et al. (1999).

The above information was imported into MSAccess as a series of six tables that are shown in the first column of Table 5-1 , labeled "All Data". The database contains a total of 28,095 records; 27,449 of which are the results of analyses of fish tissue, sediment and water samples. The remaining 646 records are contained in tables (XCLCAS and XSTN) that describe chemical identity and sample locations (Table 5-1) . Each of the 27,449 analytical data records contain the results for the analysis of a given sample and analyte. For example, if a surficial sediment sample was tested for the concentration of 15 chemicals, the database contains 15 records for that sample in the database .

For the purpose of the human health screening assessment, the site was geographically defined as the tidal Anacostia River extending from the confluence of the Northwest and Northeast Branches to the Potomac River, including Kenilworth Marsh and Kingman Lake and excluding the Washington Channel and Tidal Basin (Figure 5-1). The Tidal Basin and Washington Channel are expected to be impacted predominantly by Potomac River flow, rather than Anacostia River flow. Samples that were collected from outside of the site boundary were not included in the screening HHRA assessment. Subsurface sediment data were not used in the assessment because they may not be representative of surficial sediment, considered to be the more important exposure medium. The samples collected from within storm sewers, to the extent they could be identified, were also not included. This includes station identification names with the prefixes *'OAR'* and *'SAR'* as described in Velinsky et al. (1992). The analytical results for fish bile (Pinkney, 1999) were excluded, as this data could not be screened against the Risk Based Concentrations (RBCs) or Applicable or Relevant and Appropriate Requirements (ARARs). Table 5-1 shows the number of records in each table used in the screening database. The locations of all surficial sediment samples included in the HHRA screening database are shown in Figures 5-2 through 5-6 . Surface water and fish sampling locations are presented in Figures 5-12 and 5-13, respectively.

Two other significant issues regarding the HHRA screening database were the identification of non-detects and missing data. The data included in the HHRA screening database represent a compilation of analytical results generated by 14 different efforts. Table 5-2 provides a list of the studies that contributed data to the screening database. The NOAA (2000) database includes a table that contains the quality codes assigned to the data by the various authors (see Table 5-2 for list of citations). Data with quality codes starting with "U" were considered non-detects in the HHRA screening database. The one exception to this is the surface water data in which two-part quality codes were assigned to the combined particulate/dissolved organic data. The first part of the quality codes refers to the code assigned by Velinsky et al. (1999) to the dissolved fraction, the second part of the code refers to the code assigned to the particulate fraction.

Surface water results that were not reported by Velinsky et al. (1999) due to matrix interference were considered as *missing data* in the surface water data table ; the concentration field in the database for these records is blank and a quality code of *I* was assigned. Blanks were entered in the concentration field of the surface water data table when samples were not collected from a particular station during a sampling event. Missing data were not included in counts of samples.

5.2 SUMMARY OF THE HUMAN HEALTH RISK SCREENING ASSESSMENT DATABASE

The database includes the analytical results on a total of 356 chemicals, 141 of which are polychlorinated biphenyls (PCBs) congeners, and the following 9 physical parameters: per cent lipid in fish tissue; percent clay, silt, sand, gravel, fines and solids in sediment; and percent moisture in fish and sediment. The list of analytes varied from study to study, and sometimes between the sampling events within a given study. The total number of analytes for each media was as follows: sediment, 297 (108 PCB congeners); fish tissue, 224 (94 PCB congeners); surface water, 21.

Descriptive statistics for chemicals in each medium are provided in Tables A5-1 through A5-3 . In interpreting the summary statistics, the following limitations of the data need to be taken into account: 1) Although NOAA compiled the available information on detection limits and incorporated this information in NOAA (2000), the information in NOAA (2000) is insufficient to allow for consistent treatment of non-detects in the database. When calculating the mean and standard deviation for each chemical, non-detects were set equal to the value provided in the concentration field of the respective data tables. This value was assumed to represent the reported detection limit, however, confirmation of this against the original data was not attempted due to the size of the database. Negative concentrations were sometimes assigned to non-detects in the NOAA (2000) data tables when the detection limit was unknown. Records with negative concentrations were not included in the calculated sample means or standard deviations shown in Tables A5-1 through A5-3 , however, the number of samples includes the records assigned negative values for concentration. 2) Statistics will be tend to be biased due to non-random sampling of the site. 3) Bias can also be expected from the nonuniform geographic distribution of sampling.

Table 5-3 shows the number of samples and the detection frequencies by chemical classes. The chemical classes shown in Table 5-3 generally correspond to the classes defined in the NOAA (2000) database with the exception of the PCB data, pesticide data, and *totals* or sums of various components of analyte classes or mixtures. The NOAA (2000) database combined the pesticide and PCB data in class *(PEST-PCB)*. The PCB congener data was given its own class name. For the purpose of the human health risk screening assessment, the pesticide data was separated from the PCB data and assigned to a new chemical class (*PEST*). A chemical class called *PCB* was created for *total PCBs*. The class *total PCBs* for the sediment and fish data represents the sum of Aroclors or congeners, depending upon the study. *Total PCBs* for the water data represent the sum of the concentrations of 77 PCB congeners (Velinsky et al., 1999). Database records that represent the summation of other analytes (e.g., total PAHs, total BTEX) were not assigned to a chemical class to avoid duplicating counts of analytes from the same sample, and not included in the frequency of detection and summary statistics calculations..

5.2.1 SUMMARY OF SEDIMENT DATA

Table 5-2 lists the eight sources of information on chemicals in sediment that are included in the screening database. Information in Velinsky et al. (1992) was obtained from grab samples collected on June 18-19, 1991, from 6 locations in the tidal Anacostia River between the Pennsylvania Avenue Bridge and the Potomac River. One location was just downstream of Kingman Lake, and 5 locations were within Kingman Lake. Samples were collected from the 0–20 cm depth interval. Data supplied by Loos (1999) was derived from 4 grab samples collected near the PEPCO Benning Generating Station on October 4, 1995 and March 25, 1997 samples were collected from the top 3 cm of sediment. USFWS (1997) supplied data on composite samples collected on July 19 and August 4, 1993 from 9 locations (one composite per location) within Kenilworth Marsh. The depth from which the samples were collected is not provided in the NOAA 2000 databases or in the report (USFWS, 1997). Murphy et al. (1998) supplied data from two grab samples collected from Kenilworth Marsh in August 1996. Samples were collected from the 0–10 cm depth interval.

The other four studies that contributed data on sediment quality were not available for review at the time this report was developed. The following description of these studies is based on the information contained in the database. Baker Environmental (ND) provided data from seven locations on the northern bank of the lower Anacostia, between 11th and South Capitol Streets. The data is from samples that were collected on June 16, 1995. Baker Environmental (ND) provided data from six locations on the southeast bank of the lower Anacostia, near the confluence with the Potomac, that were sampled on July 29 and September 7, 1992. In study IDs #01 and 02, samples were collected form the 0–30.48 cm (0–12") depth interval. Data from seven locations on the northwest bank of the lower Anacostia, between 11th Street and Pennsylvania were provided by ChemWorld Environmental (1997). These samples were obtained collected on June 11, 1996 at a depth of 0–15.24 cm (0–6"). Strobel et al. (1995) provided data from one sample collected on August 26, 1990 from Kenilworth Marsh. The sampling depth is not provided in the NOAA 2000 databases (the study was not available for review).

Figures 5-2 through 5-6 illustrate the geographic distribution of sediment samples in the following 5 chemical classes: pesticides, PAHs, PCBs, acid/base/neutral extractables, and metals. Each figure shows sediment sample locations and the number of samples at each location for a given chemical class.

The geographic coverage of the sediment data, with the exception of the PCB data, tends to be similar between chemical classes, while the number of samples varies between chemical classes and varies widely between sample locations within chemical classes. The information on Aroclors is limited to the following three areas: Kenilworth Marsh; an area downstream from the 11th St Bridge; and an area near the junction with the Potomac River. Information for *total PCBs* is more evenly distributed within the site area than information concerning Aroclors (not shown in Figure 5-6), however, in most cases, only one sample was collected from each location.

The difference in the number of sediment samples collected for each chemical class, to some extent, reflects the number of analytes included in that class, which makes comparisons of the number of samples in classes difficult to interpret. However, based on patterns exhibited in Figures 5-2 through 5-6 , it is clear that the majority of the information on the concentration of chemicals in the Anacostia sediments was obtained from the following three areas: the area of the river downstream from the Pennsylvania Avenue Bridge; from Kingman Lake; and from Kenilworth Marsh. In contrast, the database contains much less information on the concentration of chemicals in sediment of the tidal Anacostia River channel, upstream from the Pennsylvania Avenue Bridge.

Figures 5-7 through 5-11 show the distribution of detection frequencies for each of the chemical classes

defined in Section 5 (ABNs, dioxins, furans, PAHs, PCBs, pesticides and metals). Note that the apparent patterns in detection frequencies may be due (at least in part) to differences in sampling and analytical methods that were used by the different studies that contributed information to the database. The list of analyses that were performed on each sample also varied from study to study which could also account for some of the variation in detection frequencies illustrated in Figures 5-7 through 5-11. However, when reviewed together with Figures 5-2 through 5-6, major data gaps in the current database become apparent. The figures also may be used to tentatively identify candidate locations for collecting additional data; which is discussed further in Section 9 - *Recommendations For Future Action.*

Figure 5-7 shows the location and detection frequency for sediment sampling stations where pesticides concentration were measured. Note the detection frequency at neighboring sampling stations tend to be similar although some variation is evident, particularly for the sampling stations located on the north bank of the Anacostia, near the 11th St Bridge. There also appears to be a difference between the sampling stations located south of the South Capitol St Bridge; the stations on the northwest bank of the river appear to have higher detection frequencies than those on the southeast bank of the river. This pattern is observed for other chemical classes as well (Figures 5-8 through 5-11). However, the detection rates shown may reflect characteristics of the river hydrodynamics as well as variation in the sampling and analytical methods that were employed by the various studies that contributed data to the database. Another potential confounding influence in the detection frequencies is the variation in the list of analytes included within each chemical class by the different studies.

Figure 5-8 shows the location and detection frequency for sediment sampling stations where polynuclear aromatic hydrocarbons (PAHs) concentration were measured The detection frequency at neighboring sample stations tend to be similar, except for the area in the vicinity of the 11th Street Bridge where the detection frequencies are highly variable, which is similar to the pattern observed for pesticides (Figure 5-7). The high variability of measured concentration of PAHs in the vicinity of the 11th Street Bridge suggests that additional samples should be collected from this area.

Figure 5-9 shows the location and detection frequency for sediment sample stations where PCB Aroclor concentrations were measured. The detection frequency for samples collected from Kenilworth Marsh is higher than the detection frequency for samples collected from the lower Anacostia. Although there are a limited number of samples from Kenilworth Marsh that were analyzed for PCB Aroclors (Figure 5-4), the pattern of detections suggests that additional samples should be collected from the upper Anacostia, as well as the lower Anacostia, to better characterize the concentrations of PCB Aroclors in the tidal Anacostia sediment.

Figure 5-10 shows the location and detection frequency for acid/base/neutral extractable chemicals (ABNs) in sediment. The spatial distribution of detection frequency is similar to those observed for pesticides, PAHs, and Aroclors. Note the high detection frequencies in the upper Anacostia and Kenilworth Marsh coincide with areas where the database contains limited information (Figure 5-5).

Figure 5-11 shows the location and detection frequency for metals in sediment. Once again, the spatial distribution of detection frequency for metals is similar to those observed for the other chemical classes. In general, the detection frequencies are high throughout the tidal Anacostia. The detection frequency for the river channel between the Pennsylvania Avenue Bridge and Watts Branch is consistently high. The database contains limited data for metals concentration is this area however (Figure 5-6), indicating a potential location where additional sampling should be considered.

5.2.2 SUMMARY OF WATER DATA

The following summary is based on data that is included in NOAA 2000; this data was derived from Velinsky et al. (1999).

As shown in Figure 5-12 , there is one sampling station located between Watts Branch and the confluence of the Northeast and Northwest Branches. Six of the eight stations are located between the 11th Street Bridge and the Potomac River.

The water data were collected from 11 stations before and after 5 rainfall events between February 25th and November 12th of 1998. Data from three of the stations, one each in the Northeast and Northwest Branches and one in the Potomac River, are not included in the database because the stations are located outside of the tidal Anacostia River site boundaries (Figure 5-1). Samples were collected from approximately 0.5 meters below the surface of the river except for one sample that was collected on February 25th from the bottom of the river at Station 6_14; the latter was analyzed for metals only.

The locations of the water sample stations included in the database are shown in Figure 5-12 . The detection frequencies for each of the chemical classes at each of the sample stations is provided in Table 5-4 . Data on water quality were obtained from a study performed by Velinsky et al. (1999) that investigated the effects of stormwater runoff on the concentrations of selected inorganic and organic contaminants in the tidal Anacostia River. The values shown in Table 5-4 include the data on total recoverable *metals* and the total *organics*. The number of samples and number of detects shown in Table 5-4 do not include the analyses for acid soluble or dissolved metals, nor do they include the dissolved and particulate analyses that were performed for the organic contaminants. Results of fractional analyses, while potentially useful for fate and transport evaluations, are not included here to avoid redundancy in data summaries.

The only analyte included in the acid/base/neutral-extractables (ABN) category is hexachlorobenzene. The metals class is comprised of arsenic, cadmium, chromium, copper, nickel, lead, zinc and mercury; the concentration of mercury was measured for the first sampling event only. The pesticide class consists of heptachlor, aldrin, trans-nonachlor, p,p'-DDE and p,p'-DDT. The PCB class represents the sum of 77 congeners.

5.2.3 SUMMARY OF FISH TISSUE DATA

Figure 5-13 shows the locations of the fish tissue samples included in the database. The detection frequencies for each chemical class and sample station are provided in Table 5-5 . Data on the concentrations of contaminants in fish tissue were obtained from 5 studies (Table 5-2).

Approximately 95% of the records in the database for fish tissue were obtained from Cummins and Velinsky (1993) and Velinsky and Cummins (1996). Information reported in these two studies is based on analyses of composite fish fillets collected in 1989-1992 and 1993-1995, respectively, from the tidal Anacostia River. The data from Cummins and Velinsky (1993) are based on the analyses of 38 composite samples that were collected from two general areas, rather than specific locations, of the river. The two areas were described as the *upper Anacostia* and *lower Anacostia*, and are shown as stations 3 and 4, respectively, in Figure 5-13 . The fish species analyzed were brown bullhead, common carp, largemouth bass, bluegill, American eel, channel catfish and pumpkinseed.

The data from Velinsky and Cummins (1996) is derived from the analyses of 20 composite fish fillet samples. The samples were also collected from two general areas of the tidal Anacostia River, the *upper*

Anacostia and *lower Anacostia*, shown as stations LA and UA, respectively, in Figure 5-13 . In addition, one sample (KM) was obtained from Kenilworth Marsh. The fish species analyzed were channel catfish, common carp, sunfish, brown bullhead and largemouth Bass.

The data from (Block, 1990 ; see the note in the ref section) were obtained from samples of *whole fish*, fillets and *carcass* collected at two locations: one near the Washington Ship Yard (LA) and one at Benning Road Bridge (BRA). Samples were collected on August 10 and 11, 1987. Two Largemouth Bass and two white catfish were collected at each site. One of each species of fish at each site was divided into a fillet and carcass sample; the remaining fish were used for the *whole fish* analyses.

The data from an additional study (Study ID #08; Table 5-2) are based on the analysis of two fillet samples, one of carp and one of striped bass, collected near the confluence of the Northeast and Northwest Branches (identified as location ANA82 in Figure 5-13) on September 25, 1995. Descriptions of the dataset that were available for the screening risk assessment did not indicate whether the fillets were composites or from single fish. Data from study ID #A1 are based on the analysis of three samples of whole fish, all white suckers, collected at approximately the same location used in Study ID #08 (although the station ID is different: 16330). It is unknown if the samples were composites or from single fish.

The amount of information on chemical concentrations in fish tissue varies widely between chemical classes and sample locations. There appears to be differences in the detection rates for PAHs and pesticides between sample stations 3/BRA and 4/LA, which indicates sample location may be important for the fish tissue data, at least for these two classes of chemicals. Based on the total number of observations by chemical class, the database contains more information on the concentration of contaminants in fish tissue samples collected between Watts Branch and the confluence with the Potomac River and less information for the area upstream of Watts Branch..

5.3 OUTSTANDING ISSUES AND RECOMMENDATIONS RELATED TO DATA SUMMARY AND COMPILATION

Several gaps and limitations in the existing database limit the usefulness of the data to support a baseline human health risk assessment.

Large sections of the river, including some important inflows and drainages, are not represented. The absence of this information may render estimates of exposure concentrations for potential receptors highly uncertain and potentially highly biased towards those geographic areas that have been more extensively sampled and to those times at which the samples were collected. The same limitations will make it extremely difficult to model chemical loadings to the tidal river.

Specific issues are outlined below.

1. One of the most challenging issues confronting use of the existing data in a human health risk assessment is the lack of adequate information on detection and sample quantitation limits for the various analyses captured in the database. This limitation has no effect on the human health screening assessment, which is based entirely on maximum concentrations detected; however, it may severely compromise the estimation of statistical parameters such as the mean concentrations and associated confidence limits which may be used in a baseline risk assessment.

2. Information on contaminant concentrations in sediment derive largely from samples collected near or downstream from the Pennsylvania Avenue Bridge. There are no sediment samples for sections of the river channel upstream of Hickey Run and relatively few sediment samples between Benning

Road Bridge and the Pennsylvania Avenue Bridge, none of which were analyzed for Aroclors. Examples of specific gaps in the sampling coverage include the following:

- X no samples for pesticides, metals or PCBs in the main channel of the river upstream from Watts Branch (Figures 5-2, 5-4, and 5-6)
- X only one sampling station for PAHs upstream of Pennsylvania Avenue Bridge (Figure 5-3)

3. There is only one water column sampling location upstream of the Independence Avenue Bridge within the tidal Anacostia River and no stations upstream from Lower Beaverdam Creek (Figure 5-12) There is no information on the concentration of dioxins, furans or PAHs in the water column.

4. Existing data were collected at various times and do not represent a random sample of the river either spatially or temporally. The data do not support a robust temporal or spatial trend analysis. Extrapolations of parameter estimates (e.g., mean exposure concentrations) over time, including extrapolations to present or future conditions will be highly uncertain.

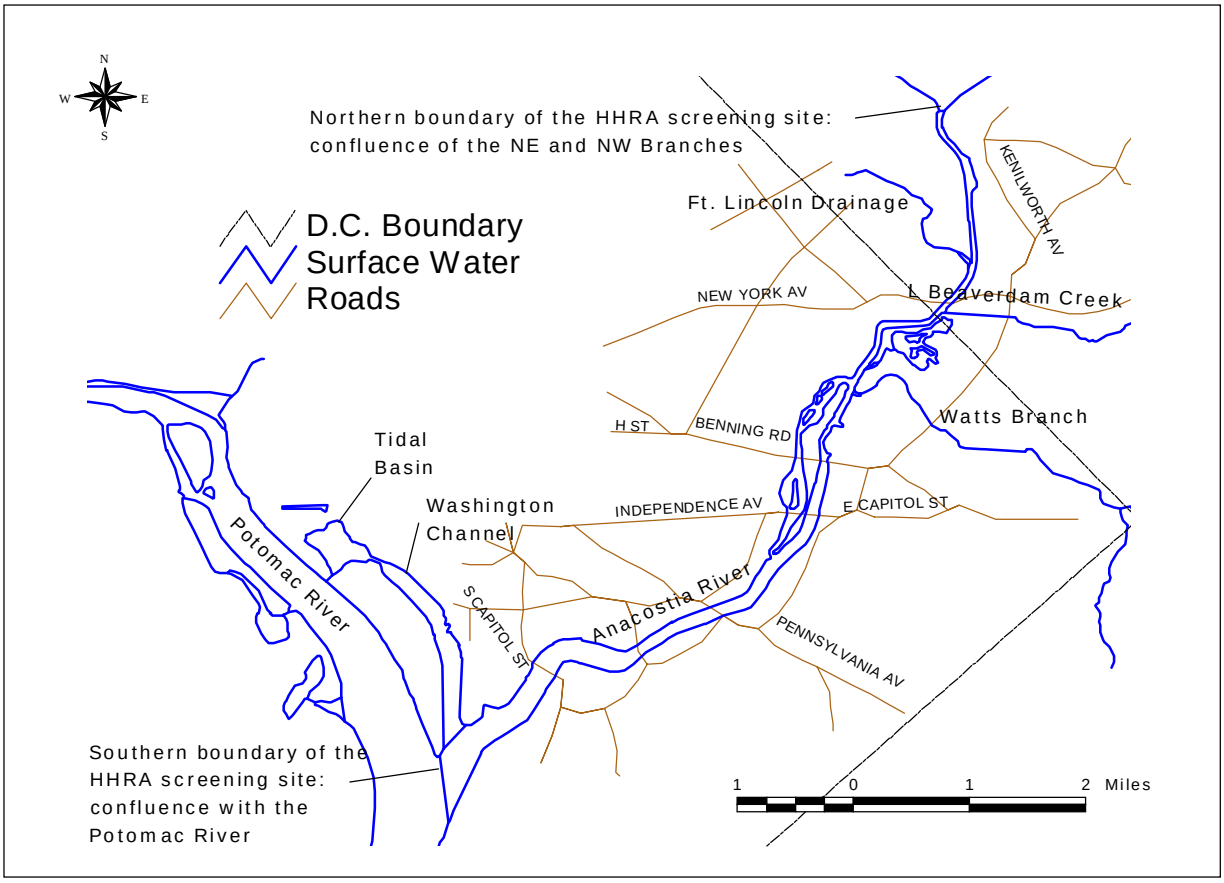


Figure 5-1. Site Map for Human Health Risk Screening Assessment. For the purpose of the human health screening assessment, the site was geographically defined as the tidal Anacostia River extending from the juncture of the Northwest and Northeast Branches to the Potomac River, including Kenilworth Marsh and Kingman Lake, and excluding the Washington Channel and Tidal Basin

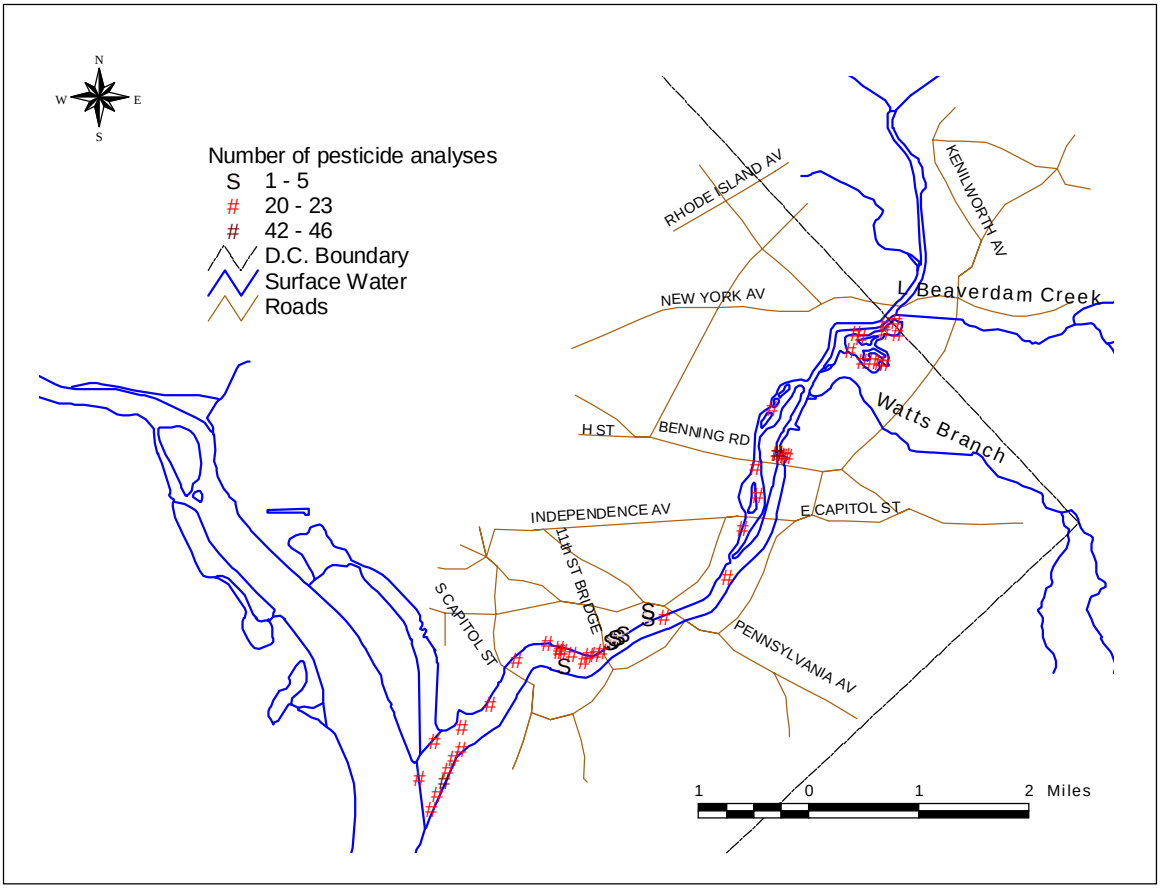


Figure 5-2. Number of Analyses for Pesticides at Sediment Sampling Stations. The figure shows a lack of samples for the tidal Anacostia River channel, upstream of Watts Branch. The large gaps in the sample number ranges shown in the legend reflect the data profile. For example, there were no sampling stations with a total number of pesticide analyses between 6 and 19.

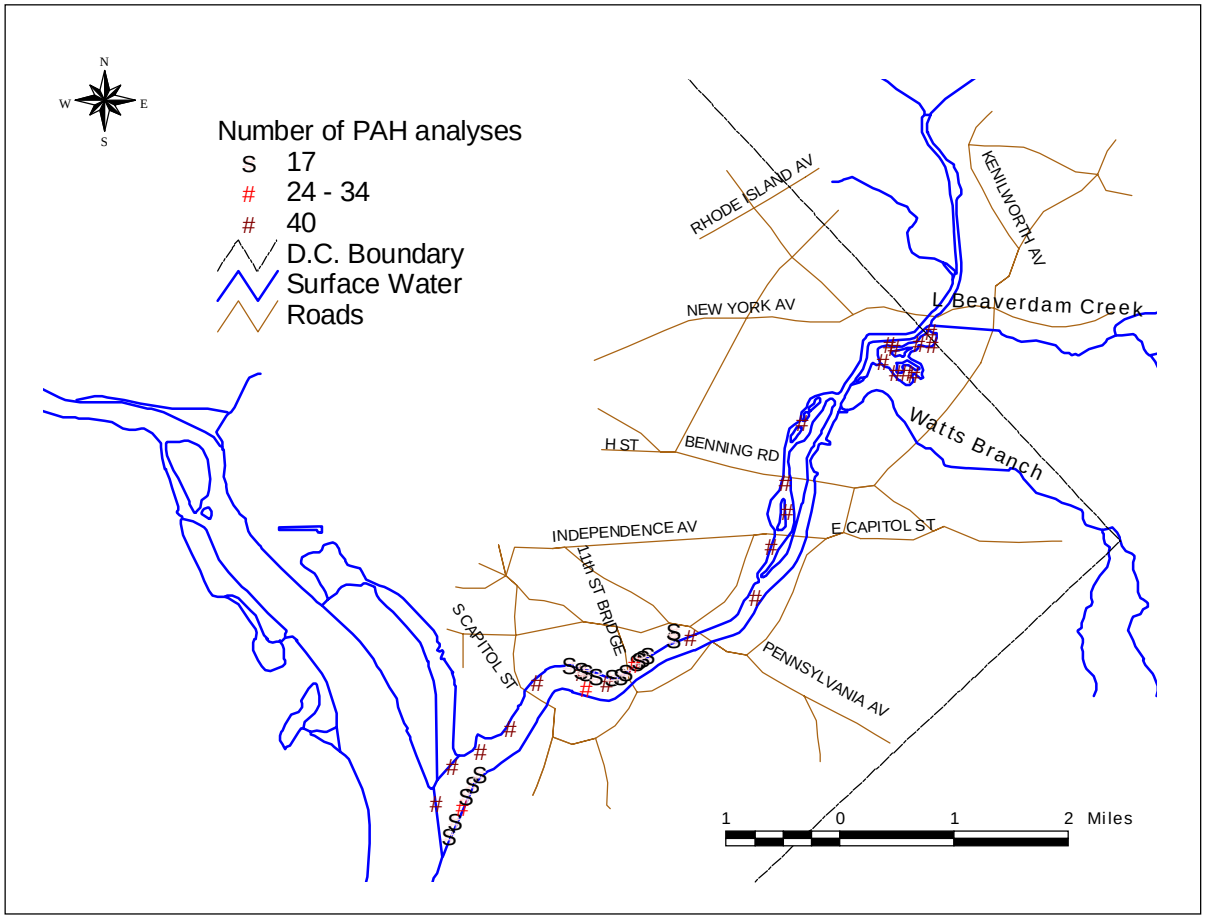


Figure 5-3. Number of Analyses for PAHs at each Sediment Sampling Station. Note that there is only one sampling station in the Anacostia River channel upstream of Pennsylvania Avenue Bridge.

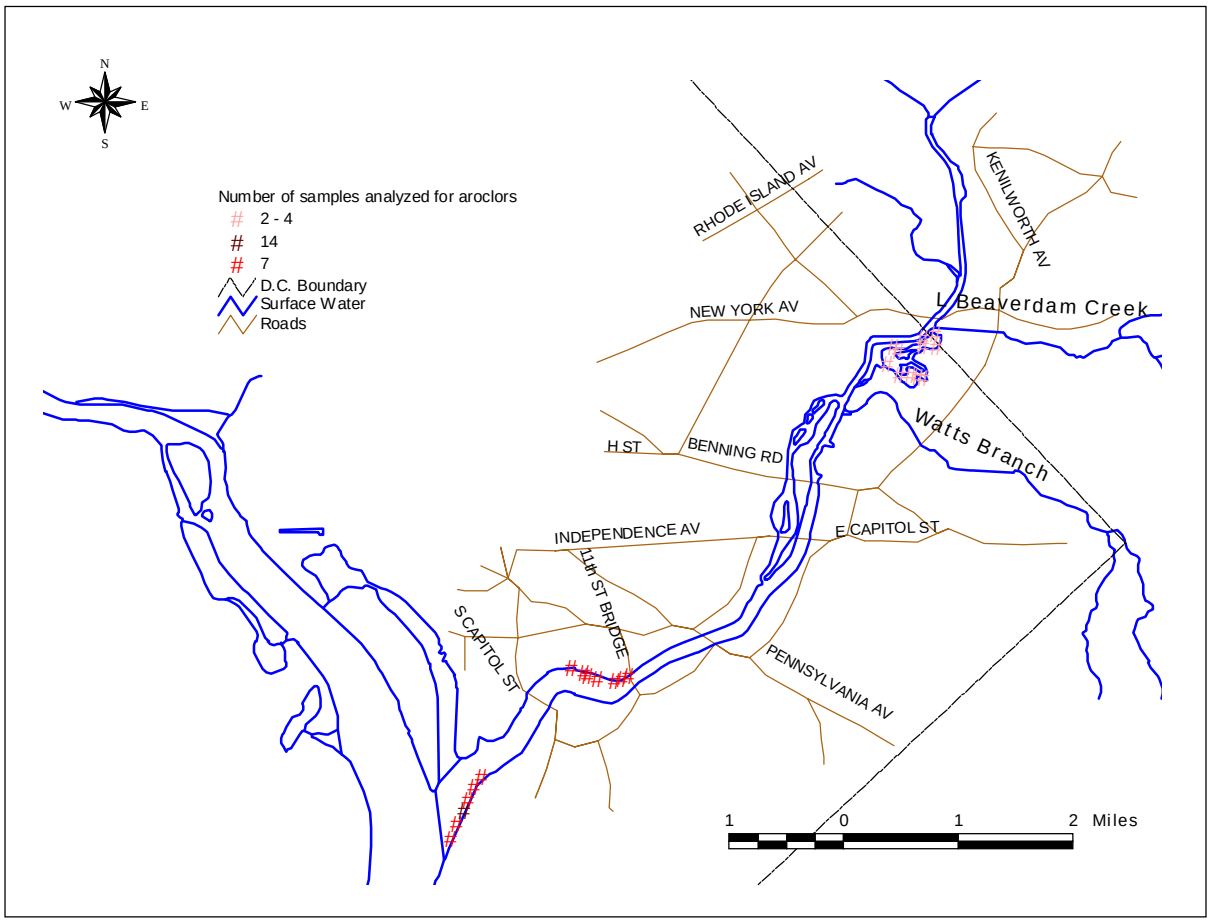


Figure 5-4. Number of Analyses for PCB Aroclors at each Sediment Sampling Station. Sediment samples collected from three areas were analyzed for aroclors: Kenilworth Marsh, near the navy shipyard and near the confluence with the Potomac.

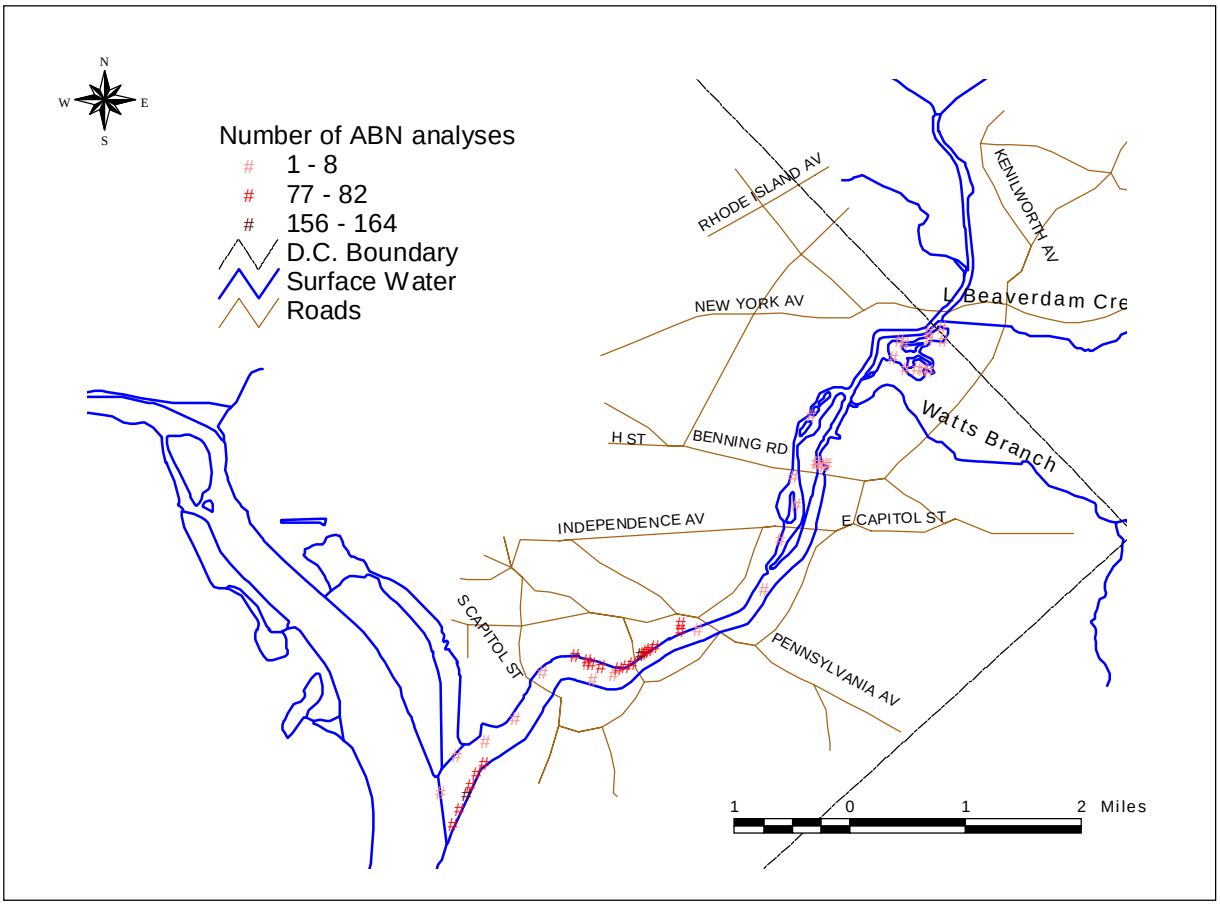


Figure 5-5. Numbers of Analyses for Acid/Base/Neutral Extractables (ABNs) at each Sediment Sampling Station. The majority of analyses have been performed on samples collected from two areas: one near the Navy shipyard and the other near the confluence with the Potomac. In contrast, there is no information in the database on concentrations of ABNs in the sediment in the river channel upstream of Watts Branch.

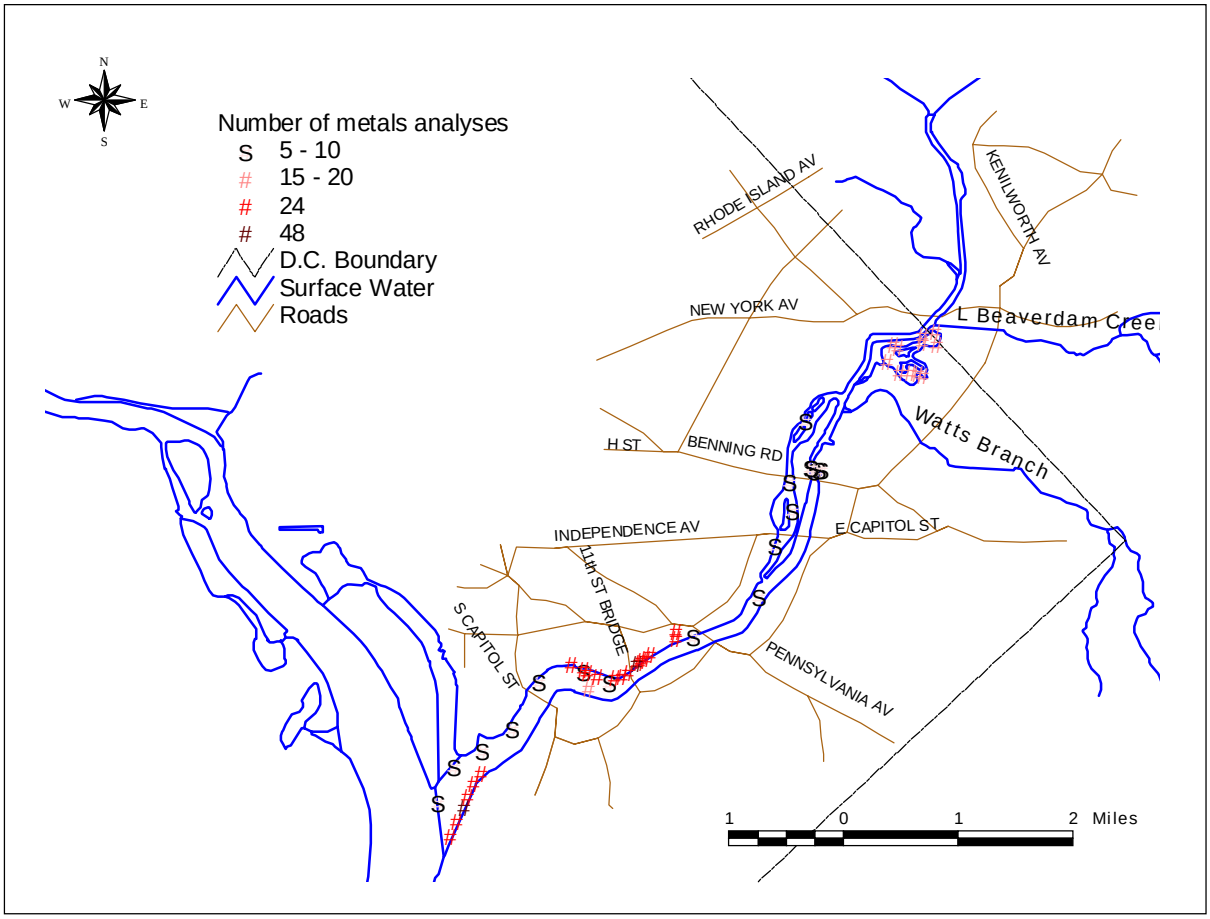


Figure 5-6. Numbers of Analyses for Metals at each Sediment Sampling Station. The majority of analyses have been performed on samples collected from three areas: near the Navy shipyard, near the confluence with the Potomac, and in Kenilworth Marsh. There is no information on the concentration of sediments in the river channel upstream of Watts Branch.

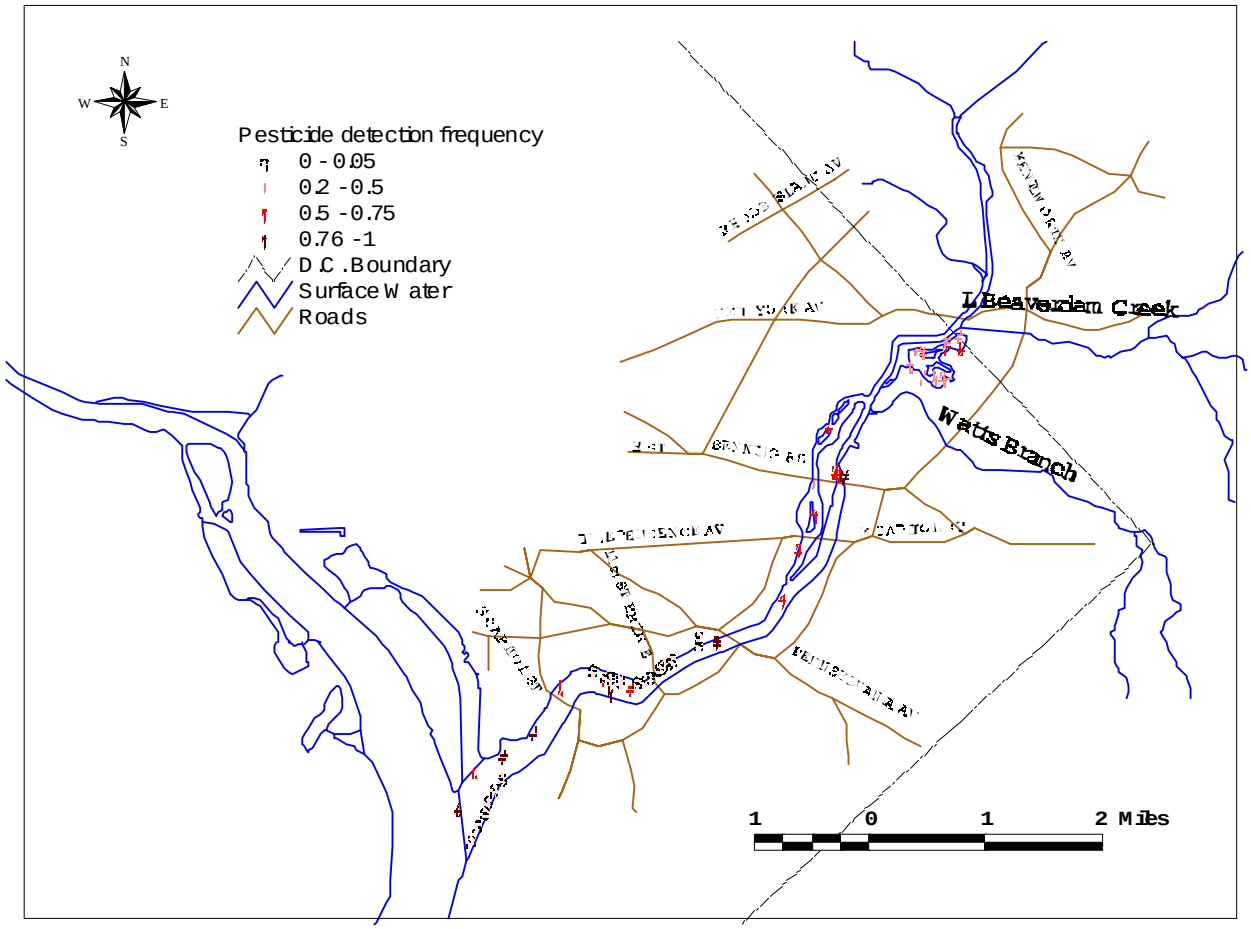


Figure 5-7. Detection Frequencies of Pesticides at Sediment Sampling Locations in the Tidal Anacostia River.

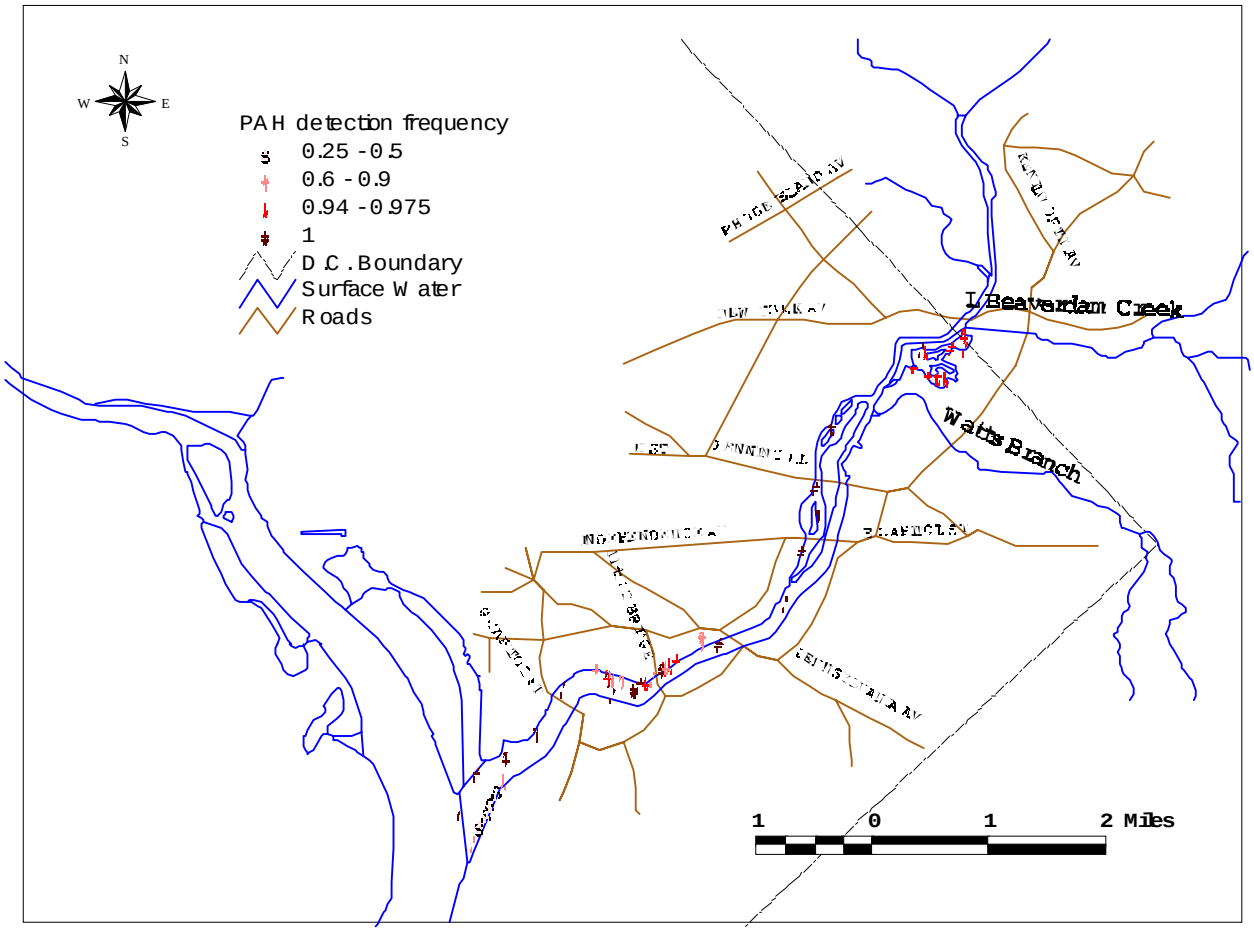


Figure 5-8. Detection Frequencies of PAHs at Sediment Sampling Locations in the Tidal Anacostia River.

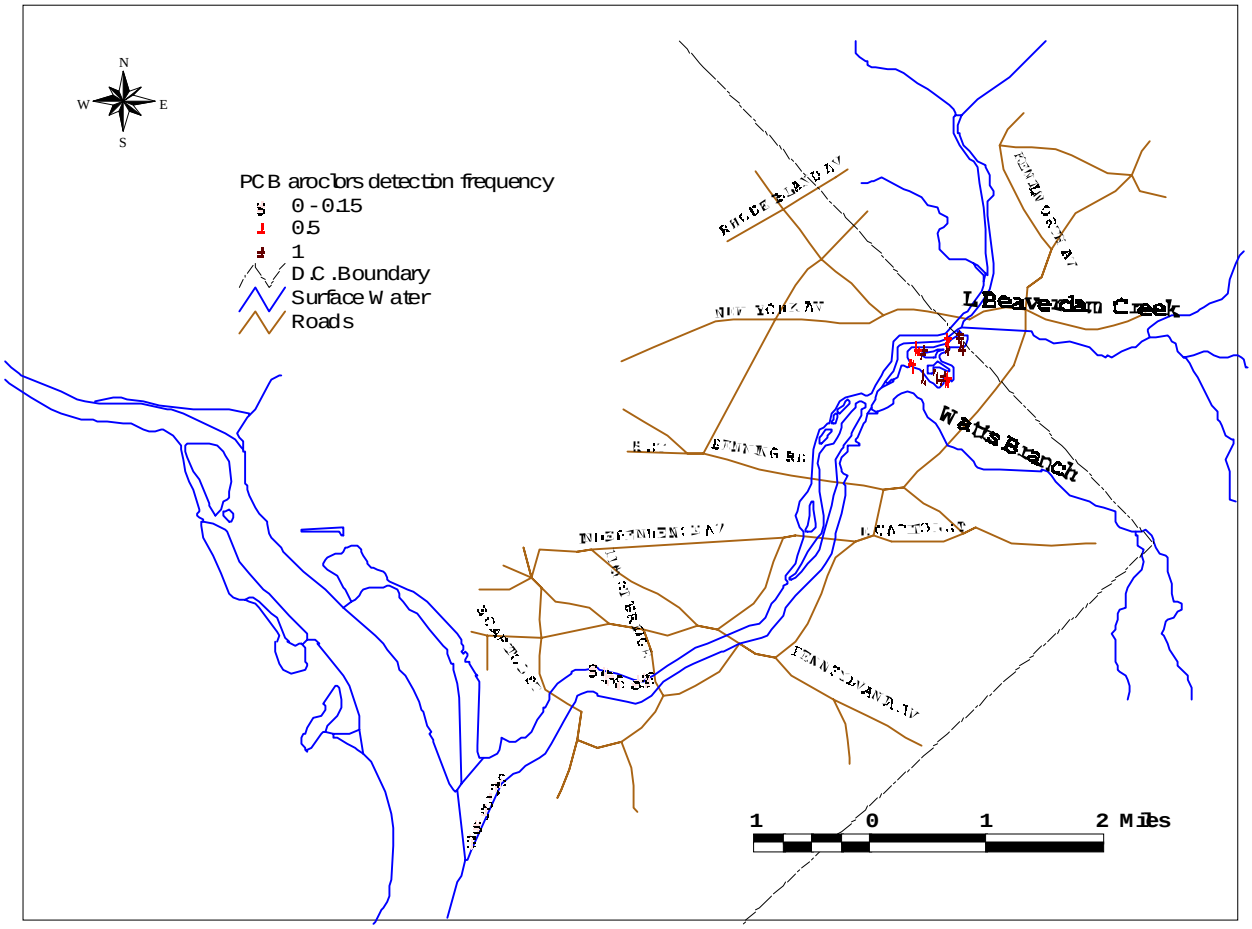


Figure 5-9. Detection Frequencies of PCB aroclors at Sediment Sampling Locations in the Tidal Anacostia River.

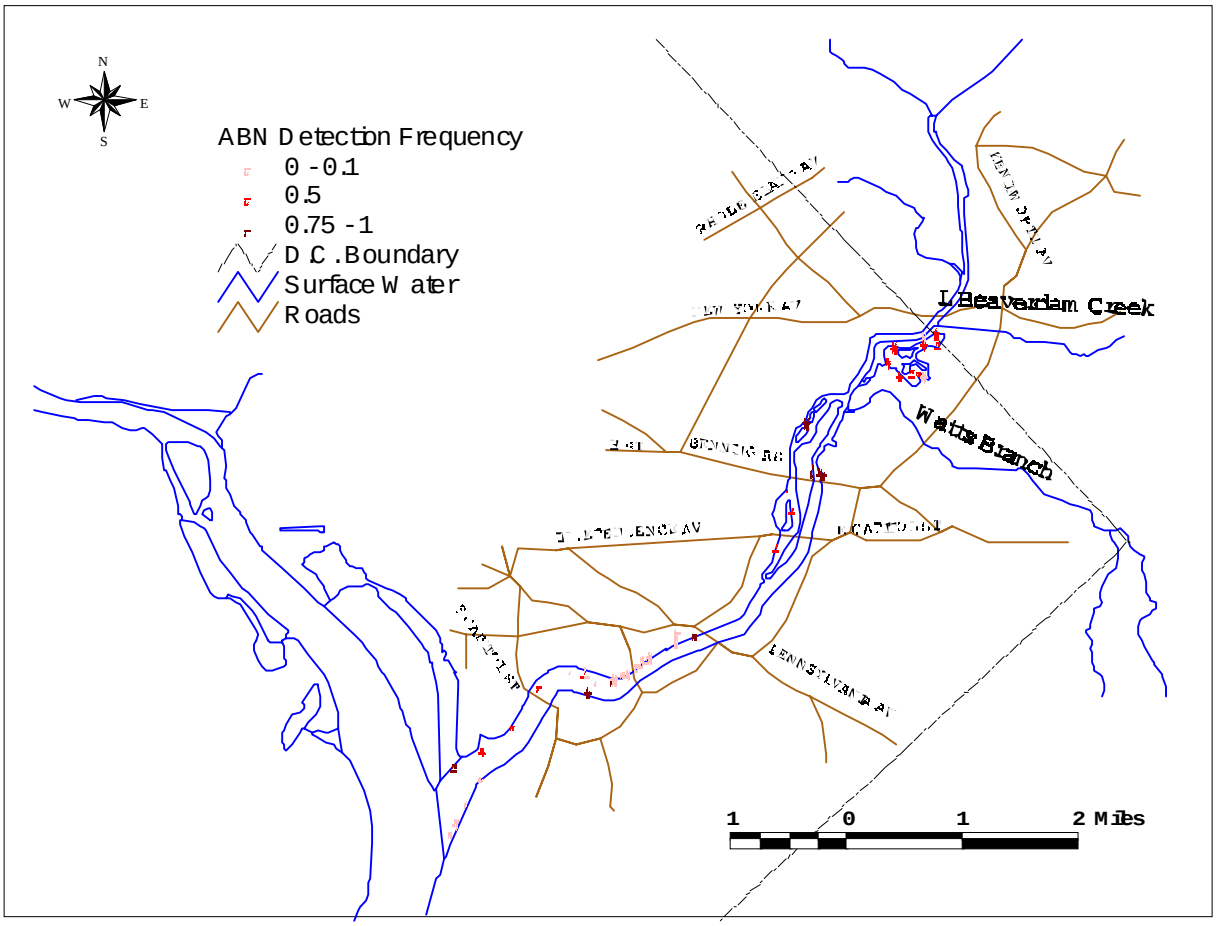


Figure 5-10. Detection Frequencies of acid/base/neutral extractable chemicals (ABNs) at Sediment Sampling Locations in the Tidal Anacostia River.

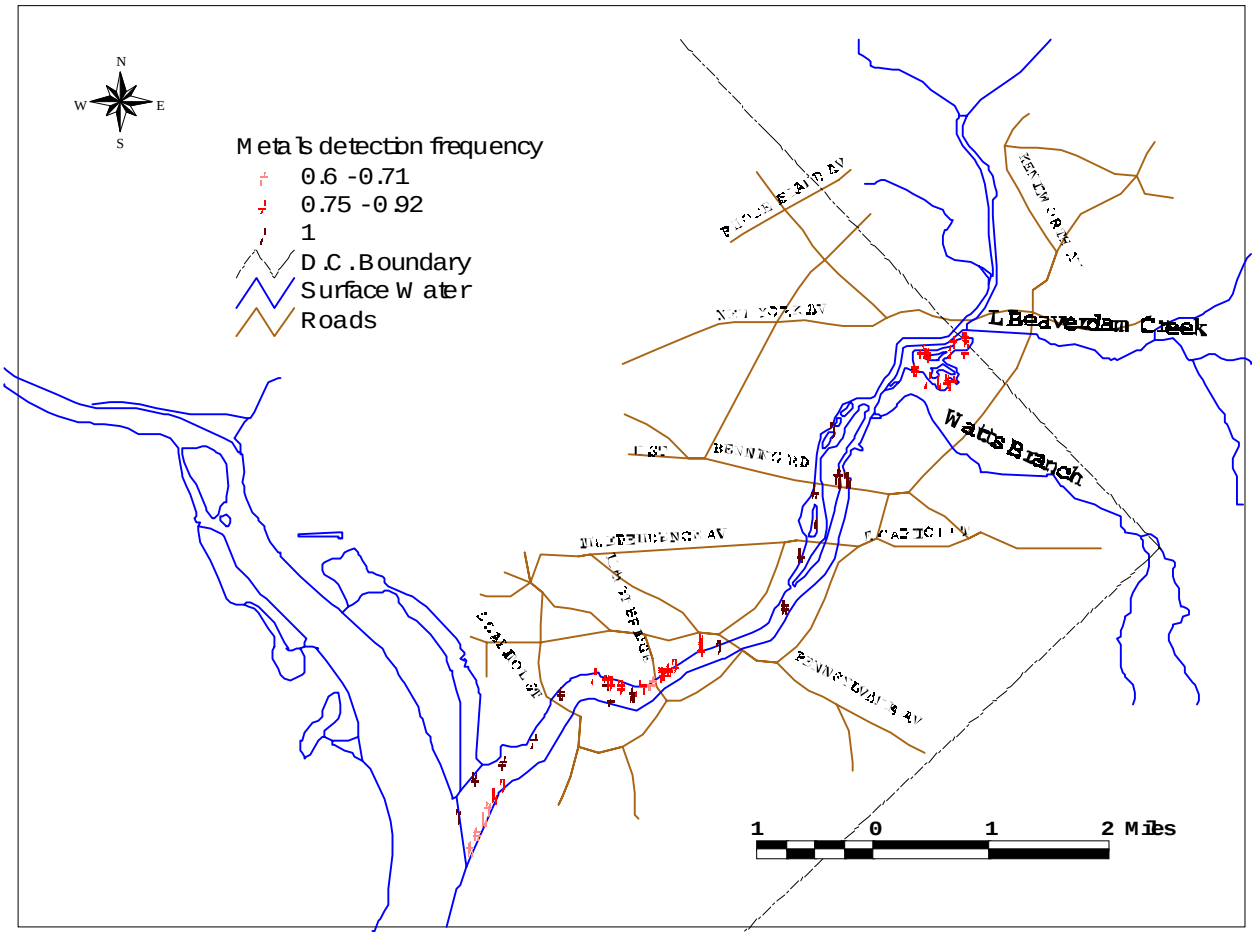


Figure 5-11. Detection Frequencies of Metals at Sediment Sampling Locations in the Tidal Anacostia River.

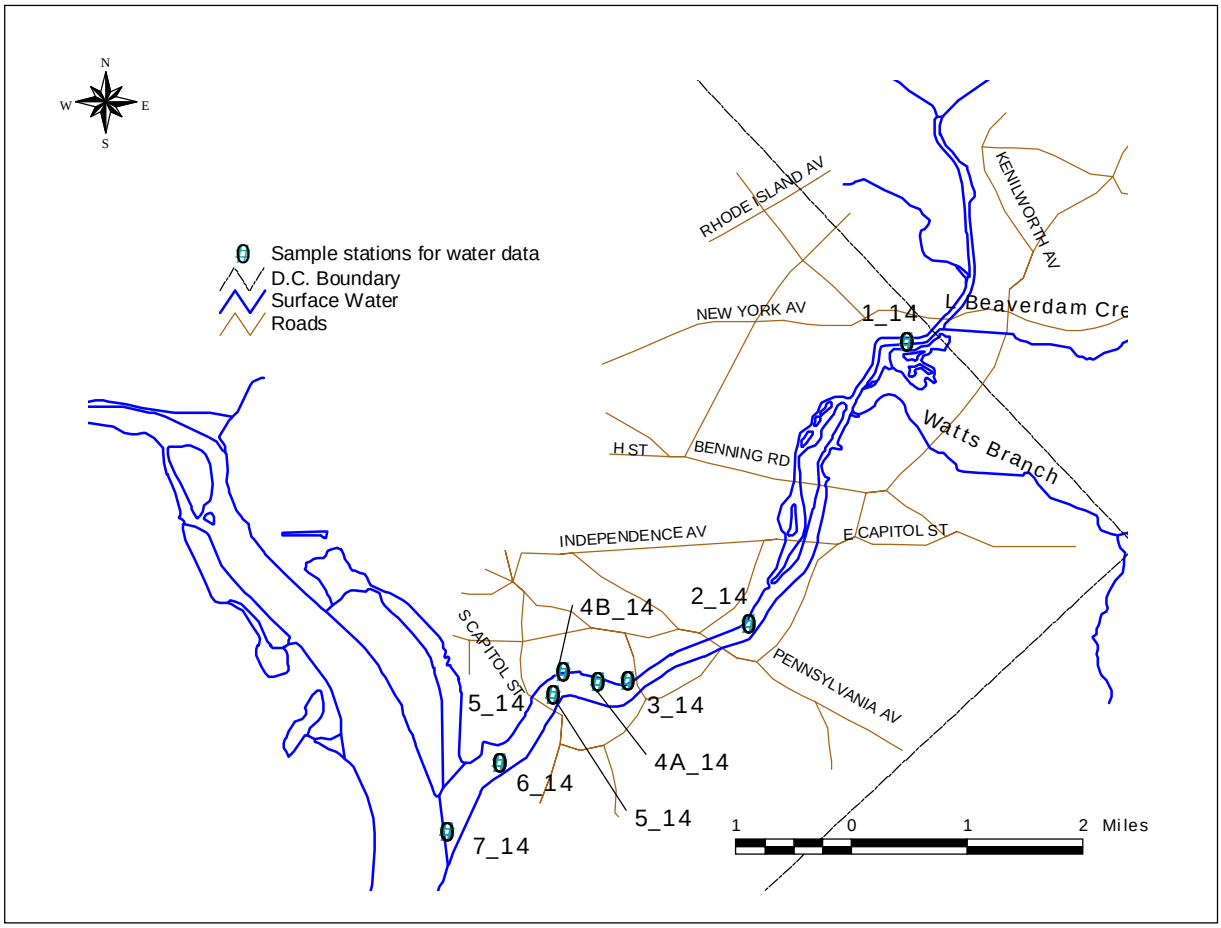


Figure 5-12. Location of Water Sampling Stations within the Tidal Anacostia. Note that there is only one station upstream of the Independence Avenue Bridge and there are no stations upstream of Lower Beaverdam Creek. The sampling stations were located in the lower Anacostia to determine the effects of stormwater runoff on water quality.

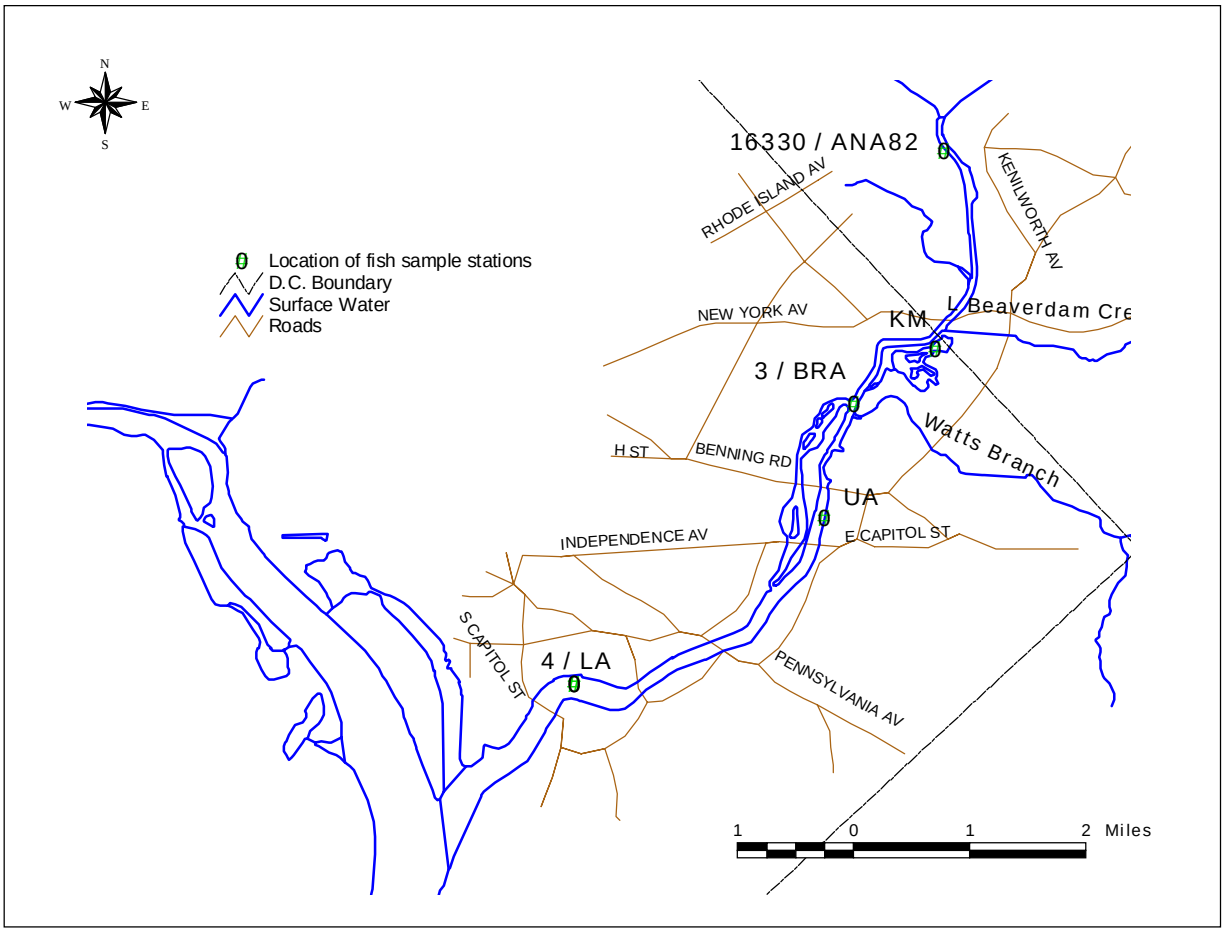


Figure 5-13. Location of Fish Tissue Sampling Stations within the Tidal Anacostia. Note that approximately 95% of the data in the database was obtained from two studies (Cummins and Velinsky 1993; Velinsky and Cummins 1996). Samples analyzed for these studies were collected from two areas: the Lower and Upper Anacostia, stations 4/LA and 3/BRA, respectively.

Table 5-1. Summary of Human Health Risk Screening Assessment Database

Table Names		No. of Records		Content Description
All Data	**Screening Data**	**All Data**	**Screening Data**	
XTISS	HHRAfish	11,009	4,932	Results of analyses of fish tissue samples
XCHEM	HHRASed	12,483	6,917	Results of analyses of surficial sediment samples
XCHEMSB	--	223	--	Results of analyses of subsurface sediment samples
Vel_ID14	HHRA_H20	3,734	2,738	Results of analyses of surface water samples
XCLAS	XCLAS	453	453	CASR numbers, chemical code names and chemical names
XSTN	XSTN	193	193	Latitude and longitude for sample locations in decimal degrees
Total Number of Records		28,095	15,233	

All Data refers to files on chemical concentrations in fish tissue, sediment and water from NOAA (2000) and Velinsky et al. (1999). Screening data refers to the subset of *All Data* that represents the tidal Anacostia as defined in Figure 5-1.

Table 5-2. Sources of Data in the Human Health Screening Database

Study ID	Study Name	Reference	Media
01	Washington Navy Yard	Baker Environmental, 1990[1]	sediment
02	Bolling AFB - SW Corner Landfill	Baker Environmental, 1990[1]	sediment
03	Additional Remedial Investigation and Feasibility Study (Phase IV) East Station Washington, D.C.	Hydro-Terra, 1999	sediment
04	1992 DC Fish Tissue Analysis for Evaluation of Human Health Risk	Cummins and Velinsky, 1993	fish

05	Organochlorine Residue/Histopathology of Fish	Block, 1990	fish
06	1995: PEPCO, Potomac Electric Power Co	Loos, 1999	sediment
08	1980-1995 Biological Tissue, Maryland	NA, 1990[1]	fish
10	Determination of Toxicity and Concentration of Contaminants In Sediment	US Fish and Wildlife Service, 1997	sediment
12	Effects of Wetland Restoration	Murphy et al., 1998	sediment
14	Effects of Stormwater Runoff on the tidal Anacostia	Velinsky et al., 1999	water
17	EMAP - Chesapeake Bay 1990	Strobel et al., 1995[1]	sediment
19	1992 Potomac and Anacostia Sediment Study	Velinsky et al.,1992	sediment
24	1993-1995 Wild Fish Tissue	Velinsky and Cummins, 1996	fish
A1	MD Dept of Natural Resources Data	NA, 1990[1]	fish

NA, Author names not available
[1]Studies were not available for review at the time this report was prepared

Table 5-3. Sample Size and Detection Frequency

Chemical Class	Number of Samples	Detection Frequency
Acid/Base/Neutral Extractables	2535	0.10
Chlorinated dibenzodioxins	126	0.92
Chlorinated dibenzofurans	180	0.98
Metals	1426	0.82
Pesticides	1899	0.46
Polycyclic Aromatic Hydrocarbons	2006	0.77
Polychlorinated Biphenyl - Aroclors	127	0.23

Chemical classes are those defined in NOAA, 2000.

Table 5-4. Detection Rates for Water Samples

Chemical Class	Sample Station ID							
	1_14	2_14	3_14	4A_14	4B_14	5_14	6_14	7_14
ABN[1]	4 / 4	5 / 5	7 / 7	1 / 1	5 / 5	7 / 7	3 / 4	5 / 5
Metal	25 / 33	55 / 59	52 / 59	30 / 33	30 / 33	49 / 59	27 / 33	41 / 59
PCB	4 / 4	5 / 5	7 / 7	1 / 1	5 / 5	7 / 7	4 / 4	5 / 5
Pesticides	12 / 20	12 / 25	18 / 35	5 / 5	16 / 25	20 / 35	8 / 20	10 / 25

Values represent the number of times a chemical in the indicated class was detected in a sample collected from the sample station divided by the total number of analyses for that chemical class at the indicated sample station. Note that there is considerable more information on metals and pesticides than there is on ABNs and PCBs.

[1]ABNs = Acid/Base/Neutrals Extractables

Table 5-5. Detection Rates for Fish Samples

Chemical Class	Station ID				
	16330 / ANA82	KM[1]	3 / BRA	UA	4 / LA
ABNs[2]	2 / 3	2 / 2	28 / 328	6 / 6	44 / 344
Dioxins	ns[3]	ns	58 / 63	ns	58 / 63
Furans	ns	ns	87 / 90	ns	89 / 90
Metals	37 / 41	7 / 12	ns	21 / 36	44 /84
PAH	ns	33 / 40	19 / 144	94 / 120	216 / 424
PCBs	3 / 3	1 / 1	14 / 15	3 / 3	22 / 22
Pesticides	15 / 45	19 / 22	94 / 234	55 / 66	226 / 388

Values represent the number of times a chemical in the indicated class was detected in samples collected from the sample station divided by the total number of analyses for that chemical class at the indicated sample station. Note that there is very little information for fish tissue, other than the concentration of metals, for samples collected north of Lower Beaverdam Creek. The apparent difference in the detection rates for PAHs and pesticides between stations 3/BRA and 4/LA indicate the fish tissue concentrations for some contaminants may vary significantly between different areas of the tidal Anacostia River.

[1]KM = Kenilworth Marsh

[2]ABNs = Acid/base/neutral-extractables

[3]NS - No Samples

REFERENCES FOR CHAPTER 5

Block, E. (1990): Organochlorine residues and histopathological examination of fish from the Potomac and Anacostia Rivers, Washington, DC. (AFO-C90-01) U.S. Fish and Wildlife Service, Environmental Contaminants Division, Annapolis, MD.

Cummins, J.D. and D.J. Velinsky. 1993. 1992 D.C. fish tissue analysis for the evaluation of human health risks. (Mid-Term Report) District of Columbia, Department of Consumer and Regulatory Affairs, Water Quality Control Branch, Water Resources Management Division, Washington, D.C. 7 pages.

Hydro-Terra, Inc. 1999. Additional Remedial Investigation and Feasibility Study (Phase IV) East Station Washington, D.C. Prepared for Washington Gas, Washington, D.C. Final March 25, 1999.

Loos, J. 1999. Contaminate concentrations observed in Anacostia River near the Benning Station after dredging. Summary results. Study No. 6. (N)

Murphy, D.R., A.E. Pinkney, R.E. Foley, P.C. McGowan, R. Li, and L. Domico. 1998. Effects of wetland restoration using Anacostia River sediments at Kenilworth Marsh. (CBFO-C98-02) U.S. Fish and Wildlife Service, Annapolis, MD. 200 pages.

NOAA. 2000. Anacostia River Watershed Database and Mapping Project. Release 1. National Oceanic and Atmospheric Administration, Office of Response and Restoration, Coastal Protection and Restoration Division.

Pinkney, G. 1999. Investigation of polynuclear aromatic hydrocarbon (PAH) and polychlorinated biphenyl (PCB) contamination at the Mason Neck National Wildlife Refuge complex: Linkages to tumors in Brown Bullhead and analysis of cytochrome P450 in Great Blue Heron. Department of the Interior, U.S. Fish and Wildlife Service, Region 5, Environmental Contaminants Program On-Refuge Investigations Sub-Activity.

U.S. Fish and Wildlife Service (1990): Organochlorine residues and histopathological examination of fish from the Potomac and Anacostia Rivers, Washington, DC. (AFO-C90-01) U.S. Fish and Wildlife Service, Environmental Contaminants Division, Annapolis, MD. 30 pages.
Note: The NOAA database lists Block, E. as the author for this document; her name appears on the cover page of the document, for what that is worth.

U.S. Fish and Wildlife Service (1997): Data report: Determination of toxicity and concentrations of inorganic and organic contaminants in sediments used to restore Kenilworth Marsh, Washington, DC. (CBFO-C97-02) U.S. Fish and Wildlife Service, Branch of Water Quality and Environmental Contaminants, Annapolis, MD. 100 pages.

Velinsky, D.J., C. Haywood, T.L. Wade, and E. Reinharz. 1992. Sediment contamination studies of the Potomac and Anacostia Rivers around the District of Columbia. (Final Report; ICPRB Report No. 92-2) Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Hygiene Branch, Washington, DC. 147 pages.

Velinsky, D.J. and J.D. Cummins. 1996. Distribution of chemicals in 1993-95 wild fish species in the District of Columbia. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Water Resources Management Division, Washington, DC.

Velinsky, D.J., G.F. Riedel, and G. Foster. 1999. Effects of stormwater runoff on the water quality of the tidal Anacostia River. Final Report 8PO-331-NAEX ed. U.S. EPA, Region III, Philadelphia. 132 pages.

6. HUMAN HEALTH RISK SCREENING

6.1 CONCEPTUAL CONSTITUENT INFLUX AND TRANSPORT MODEL

A conceptual site model (CSM) describes the processes that link sources of contamination at a site to exposures of human or ecological receptors. Ideally, the model defines the inputs of constituents to a site, the physical and chemical processes that result in transport of the constituents into environmental media to which human or ecological receptors may come into contact, and identifies the receptors that are likely to be impacted by exposure to these media. In its mature form, the model provides a basis for planning of data collection and evaluation needed to support risk assessments and remedial actions. In the early stages of development, the CSM identifies all *potential* links between sources and receptors, which subsequently can be evaluated for their plausibility and relevance with further data collection and analysis. Inputs, fate and transport processes, and exposure scenarios that are subsequently determined to be implausible or of negligible importance can be eliminated based on sufficient evidence.

The draft CSM described here is in the preliminary stages of development. It includes all of the potential inputs and exposure pathways of potential receptors. At this stage, the model does not attempt to quantify the relative importance of the various processes and pathways. The model is generic with respect to constituents. As constituents differ in the degree to which they may be affected by various fate and transport processes, at some point in the risk assessment process individual chemical-specific or chemical class-specific models may need to be developed and evaluated.

The draft CSM for human exposures at the tidal Anacostia River is shown in the attached Figures 6-1 and 6-2 . For the purposes of this screening risk assessment, and at the direction of the Alliance, the tidal Anacostia River is defined as the river proper, including the tideplain and floodplain, extending from the confluence of the Northeast and Northwest Branches to the confluence with the Potomac River, as well as the Kenilworth Marsh and Kingman Lake embayments. It should be noted that this geographic constraint needs to be evaluated in terms of whether or not the contamination of the tidal Anacostia River can be adequately understood for the RI/FS process without considering inputs of contamination to the greater Anacostia Watershed. The draft CSM identifies potential exposure pathways linking sources with human receptors at the tidal Anacostia River. The model includes three components: *input pathways*, *tidal Anacostia River pathways*, and *exposure scenarios*, which are illustrated in the left, middle and right sections of Figure 6-1 .

The *input pathways* component identifies the transfer mechanisms by which15 potential *antecedent media* enter the tidal Anacostia River as surface water, including suspended solids, which is referred to in Figure 6-1 as *contributed media*. *Antecedent media* is defined here as the various media (e.g., groundwater, Potomac River sediments) that potentially contribute chemical constituents to the Tidal Anacostia River (see Figure 6-1). In a complete CSM for a given chemical or chemical class, the relative contribution of each of the potential antecedent media to total surface water and suspended solids would be represented quantitatively. In the screening assessment, this will be evaluated based on available information, and information gaps will be identified for further study. Information concerning analyte concentrations in antecedent media could be used in the forthcoming baseline risk assessment to identify potential sources of contamination.

The *tidal Anacostia River pathways* component identifies the transport mechanisms that operate within the tidal Anacostia River by which chemicals entering the system in surface water may be distributed to,

and move between, the various media to which receptors may come into contact (*potential contact media*). Constituents may exit or be effectively isolated from contact media through the processes of chemical transformation, air movement, burial in deep riverbed sediments, or transport in surface water outflow to the Potomac River.

The *exposure scenario* component of the model identifies the potential scenarios by which humans may be exposed to contact media and indicates the current state of knowledge regarding the completeness of the exposure pathway for each scenario. Potential exposure scenarios are identified in terms of contact medium, exposure route, and receptor exposure category. Figure 6-2 provides a more detailed exposure scenario component of the CSM in that it identifies specific human receptor activities that would fall into each exposure category. The pathway evaluations shown in Figures 6-1 and 6-2 are based on review of the available information and may require modifications based on additional information provided from subsequent data collection efforts. In particular, currently available information does not support a quantitative analysis of the relative magnitude by which each transfer mechanism contributes to exposures. A more quantitative model is desirable for evaluating remediation strategies. For example, it is our understanding, based on comments from U.S. EPA, that there are no active domestic or commercial wells in the tidal Anacostia area that are used for residential tap or drinking water, therefore, the groundwater pathways are indicated as incomplete in Figures 6-1 and 6-2 .

Figure 6-1. Conceptual Site Model for Human Health Risk Screening Assessment of the Tidal Anacostia .

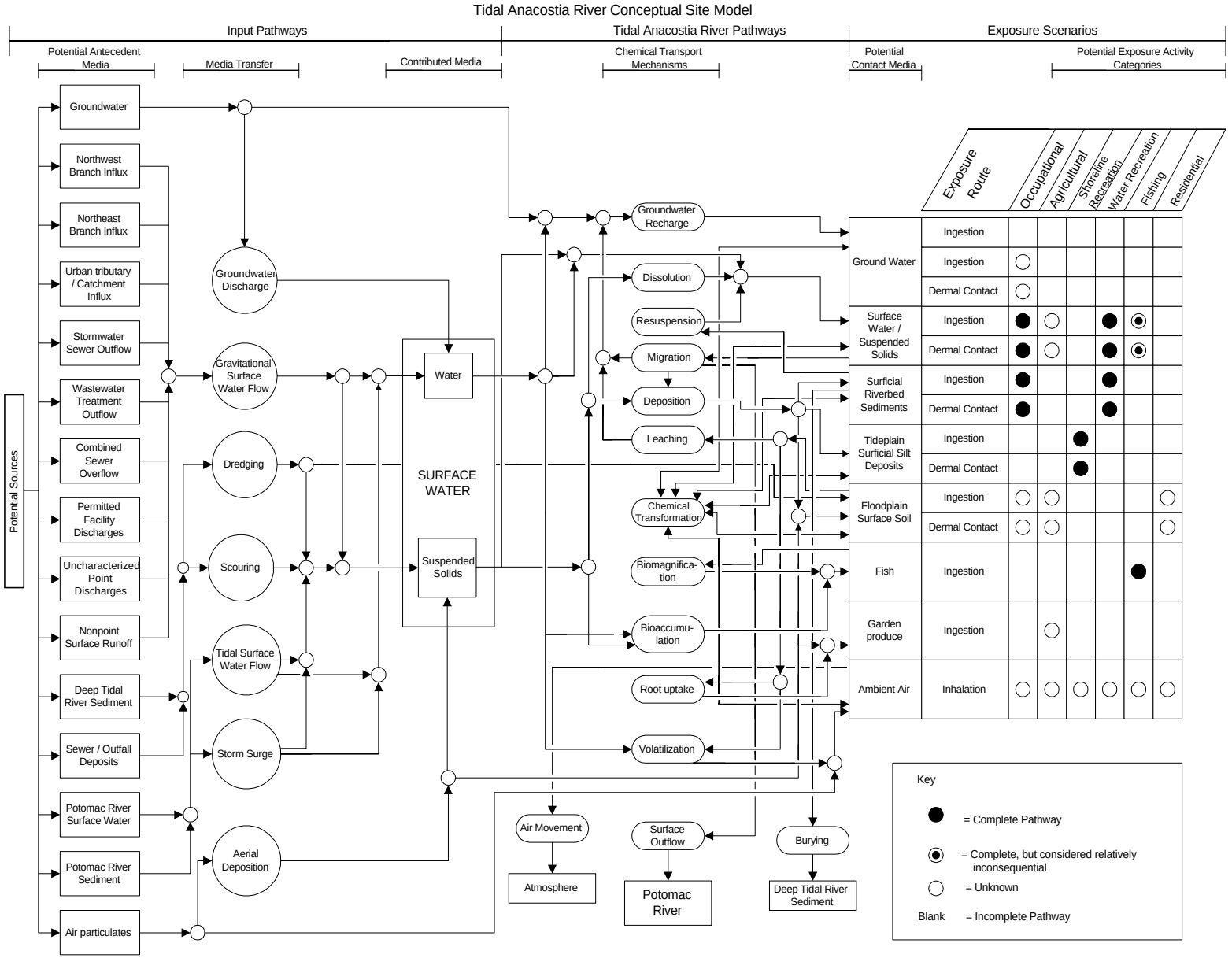


Figure 6-2. Potential Human Exposure Scenarios and Receptors Associated with the Tidal Anacostia River

Contact Media	Exposure Route	Occupational: Construction	Occupational: Dredging	Occupational: Fishing	Agricultural: Gardening	Shoreline Recreational: Picnicking, Hiking, Biking	Water Recreational: Boating	Water Recreational: Skiing, Swimming, Wading	Fishing: Recreational	Fishing: Subsistence	Residential: Showering	Residential: Consuming Drinking Water	Residential: Yard Play
Ground Water	Inhalation												
	Ingestion	○											
	Dermal Contact	○											
Surface Water / Suspended Solids	Ingestion		●	●	○		●	●	◉	◉			
	Dermal Contact		●	●	○		●	●	◉	◉			
Surficial Riverbed Sediments	Ingestion		●					●					
	Dermal Contact		●					●					
Tideplain Surficial Silt Deposits	Ingestion	○				●							
	Dermal Contact	○				●							
Floodplain Surface Soil	Ingestion	○			○	○							○
	Dermal Contact	○			○	○							○
Fish	Ingestion								●	●			
Garden Produce	Ingestion				○								
Ambient Air	Inhalation	○	○	○	○	○	○	○	○	○			○

Key — Pathway Analysis

● = Complete Pathway

◉ = Complete, but considered relatively inconsequential

○ = Unknown

Blank = Incomplete

6.2 SCREENING LEVEL ASSESSMENT

6.2.1 DECISION FRAMEWORK FOR SCREENING AND DATA INPUTS

The primary purpose of the screening level human health risk assessment is to categorize chemicals in the tidal Anacostia River with respect to their potential for adversely affecting human health. The chemicals that were considered in the assessment were all chemicals identified in the Anacostia River Watershed Database and Mapping Project (NOAA, 2000) (Table A6-1) (see Section 5- *Existing Data Summary/Compilation* for additional information on the database). The exposure pathways and receptors that were considered included those indicated in the in the CSM (Figures 6-1 and 6-2)

Figure 6-3 illustrates the decision framework used to screen chemicals. The framework sorts chemical into five categories: 1) Chemicals of Potential Concern (COPC), 2) Not COPC, 3) Insufficient Information Related to Exposure; 4) Insufficient Information Related to Toxicity; or 5) Not Detected in Watershed.

Category 1 (COPC) includes chemicals detected in the tidal Anacostia River whose maximum concentration exceeded an RBC or an Applicable or Relevant and Appropriate Requirement (ARAR), and exceeded the expected site background concentration, if a site background estimate was available. The database does not include samples that would represent local background (i.e., concentrations of chemicals in sediment, water column and aquatic biota that would be expected in the absence of potential inputs to the tidal Anacostia River), therefore, the background component of the screen could not be conducted with available data.

Category 2 (Not COPC) would include any chemicals for which we can be reasonably certain do not pose an unacceptable risk, given the information available at this time. That is, there is sufficient information on exposure and toxicity of each Category 2 chemical to satisfy the screening requirements, and the maximum chemical concentration does not exceed an RBC or ARAR (a background screen could not be conducted).

Category 3 (Insufficient Information Related to Exposure) includes chemicals for which sampling was considered to be inadequate to interpret the maximum concentration reported in terms of potential risks, even though the reported maximum concentration did not exceed an RBC or ARAR. Category 3 also includes chemicals that were not detected in the tidal Anacostia River and for which sampling was considered to be inadequate. Adequacy of sampling was evaluated in terms both the number samples and geographic distribution of sampling within the tidal Anacostia River. The screening criteria for Category 3 was a minimum of three samples representing the upper, middle and lower regions of the tidal Anacostia River, including areas immediately downstream from the major inflows and catchments: 1) Northeast and Northwest Branches; 2) Fort Lincoln Drainage; 3) Lower Beaver Dam Creek; 4) Hickey Run and Watts Branch; 5) Washington Channel area; 6) Kenilworth Marsh; and 7) Kingman Lake. Category 3 would also include any chemicals detected in the watershed outside of the tidal Anacostia River (e.g., the Northeast and Northwest Branches) that were not evaluated in the tidal Anacostia River. Chemicals in Category 3 warrant further characterization with respect to their concentrations and distributions within the tidal Anacostia River before a determination can be made as to whether or not they are COPCs.

Category 4 (Insufficient Information Related to Toxicity) includes detected chemicals for which there were no applicable RBCs or ARARs and, therefore, a determination cannot be made as to whether or not they are COPCs. Should toxicological information become available on any of these chemicals be found sufficient to support the development of provisional RBCs, their status could be reevaluated.

Category 5 (Not Detected in Watershed) would apply to any chemicals that were not evaluated in the tidal Anacostia River, however, they were evaluated in other parts of the watershed and were not detected.

These chemicals were included in a separate category because there is no evidence that they would be transported into the tidal Anacostia River from other parts of the watershed; therefore, their possible occurrence in the tidal Anacostia River would be from sources specific to the tidal Anacostia River. The need for further evaluation of the chemicals in Category 5 would be based, in part, on considering the likelihood of the existence of such potential sources. The complement of Category 5 are chemicals that were not evaluated in the tidal Anacostia River but were detected in other parts of the watershed; these were included in Category 3 (Insufficient Information Related to Exposure).

6.2.2 RISK BASED CONCENTRATIONS (RBCS)

Data were available on chemical concentrations in three environmental media in the tidal Anacostia River: river sediment, river surface water and river fish tissue. Chemicals in river surficial sediment were screened against RBCs for soil for a *commercial/industrial exposure scenario*, using the relevant RBCs developed by U.S. EPA Region 3 (U.S. EPA, 1999). The RBCs represent the chronic exposure concentration that would result in a non-cancer health risk equivalent to a Hazard Quotient (HQ) of 0.1 or a Cancer Risk of (CR) of 10^{-6}. The exposure factors integrated into the soil RBCs are summarized in Table 6-1 . These factors, when used with the maximum sediment concentration and risk criteria of HQ #0.1 and CR #10^{-6}, introduce an appropriately *conservative* (health protective) *bias* into the sediment screening assessment for the following reasons: 1) potential receptors can be expected to be exposed to an average concentration less than the maximum concentration used in the screening assessment; 2) the risk criteria represent risks that are generally considered acceptable for environmental exposures to chemicals; and 3) the soil ingestion rate of 100 mg/d (and other exposure factors) represents a reasonable maximum exposure (RME) estimate for ingestion of soil and probably overestimates the RME estimate for river sediment.

Chemicals in surface water were screened against RBCs for residential tap water (U.S. EPA, 1999). The exposure factors integrated into the tap water RBCs are summarized in Table 6-2 . The factors account for ingestion of tap water as well as inhalation of volatile chemicals during the use of tap water (e.g., showering). For the same reasons noted in reference to the soil RBCs, the tap water RBCs introduce a *conservative bias* into the screening assessment when applied to the surface water exposure scenario for the tidal Anacostia River. In particular, the tap water ingestion rates would be expected to substantially overestimate RME estimates for ingestion of surface water because Anacostia River water is not used for tap water in the region. Residences in the tidal Anacostia River area are supplied with municipal water and there is no documented use of ground water (e.g., wells) to supply household taps.

Chemicals in fish flesh were screened against RBCs for edible fish (U.S. EPA, 1999), using the exposure factors presented in Table 6-3 . The RBCs for fish are based on wet weight fillet, however, the screening assessment is based on whole fish as well as fillets; this allowed inclusion of all of the fish tissue data in the assessment. The use of the whole fish data may result in a (health-protective) *conservative bias* in the screening assessment since chemicals in the parts of the fish that may not be ingested (e.g., subcutaneous fat and skeleton), and which may have higher concentrations in these tissues than in lean muscle (e.g., PCBs, lead), are included in the assessment. The fish tissue concentrations in the database are based on composite samples. Maximum concentrations determined from composite samples will tend to be biased low due to the *'averaging'* or *'smoothing'* effect that results from combining samples with high concentrations of contaminants with samples that have lower concentrations. However, the use of the maximum concentration as the RME exposure concentration may offset the low bias in the exposure concentration introduced by compositing samples.

Lead in river sediment was screened against a soil RBC of 800 mg/kg (ppm). The basis for the RBC is the U.S. Interim Adult Lead Methodology and the relevant exposure factors are provided in Table 6-4 (U.S. EPA, 1996). The methodology predicts that chronic exposure of women of childbearing age to

800 mg/kg lead for 219 days per year would result in a 95th percentile fetal blood lead concentration that would not exceed 10 :g/dL. Lead in river water was screened against the ARAR of 15 :g/L, the current U.S. EPA Maximum Contaminant Level for drinking water (U.S. EPA, 1991).

A Toxicity Equivalency Factor (TEF) approach was used to screen *dioxin-like* congeners of chlorinated dibenzo-*p*-dioxins (CDDs) and chlorinated dibenzofurans (CDFs). The maximum concentration of each congener was multiplied by the respective TEF and the resulting product was screened against the RBC for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) (U.S. EPA, 1999). Separate screening assessments were conducted using TEFs recommended by U.S. EPA (1989) and the World Health Organization (WHO) (Van den Berg et al., 1998) (Table 6-5). However, the U.S. EPA TEFs were used in the final screening because they yielded more conservative estimates of 2,3,7,8-TCDD toxicity equivalent RBCs than did the WHO TEFs.

Aroclor mixtures of polychlorinated biphenyls (PCBs) were screened against respective RBCs (U.S. EPA, 1999). Congeners of PCBs were not screened, as per general procedures for screening assessments conducted by U.S. EPA Region 3. Polycyclic aromatic hydrocarbons (PAHs) were screened individually based on RBC values reported in U.S. EPA (1999).

6.2.3 APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARS)

Chemicals were also screened against medium-specific ARARs and those chemicals for which the maximum detected concentration exceeded an ARAR were also considered COPCs (Category 1, Figure 6-3). ARARs used in the screening assessment are provided in Tables A6-2,3.

Water samples collected from the Tidal Anacostia river were compared to the D.C. surface water quality criteria (DCSWQC) (GDC 1994). If no criterion was provided for a particular chemical in the DCSWQC, the criterion for that chemical was obtained from the National Recommended Water Quality Criteria (NRWQC) (U.S. EPA, 1998), if one was provided. Finally, if no criterion for a particular chemical was provided in the DCSWQC or the NRWQC, the criterion for that chemical was obtained from the Maryland Water Surface Water Quality Criteria (MDE 2000), if one existed. In all cases, the criteria obtained from the above three regulations were developed to be protective of human health.

Fish tissue samples were compared to cancer and noncancer risk-based concentrations established for fish tissue by the U.S. EPA and to U.S. Food and Drug Administration (U.S. FDA) tolerance, action or guidance levels that are provided in Table D-1 of the National Sediment Quality Survey (U.S. EPA 1997). The EPA values in Table D-1 were adjusted to correspond to a lifetime cancer risk of 1E-06 or a hazard quotient of 0.1. When more than one screening value was provided for a given chemical, the lowest was used.

The sediment screening values provided in Table D-1 of the National Sediment Quality Survey (U.S. EPA, 1997) were considered as potential ARARs but were not used because the values provided in Table D-1 were developed to be protective of aquatic organisms rather than humans. The generic soil screening levels (SSLs) provided in the U.S. EPA Soil Screening Guidance Document (U.S. EPA 1996) were also considered as potential ARARs. The SSLs were not used because they assume a residential exposure scenario which is not consistent with the Region 3 RBCs for soil (U.S. EPA, 1999) (which assume an industrial exposure scenario) that were used in the human health screening assessment.

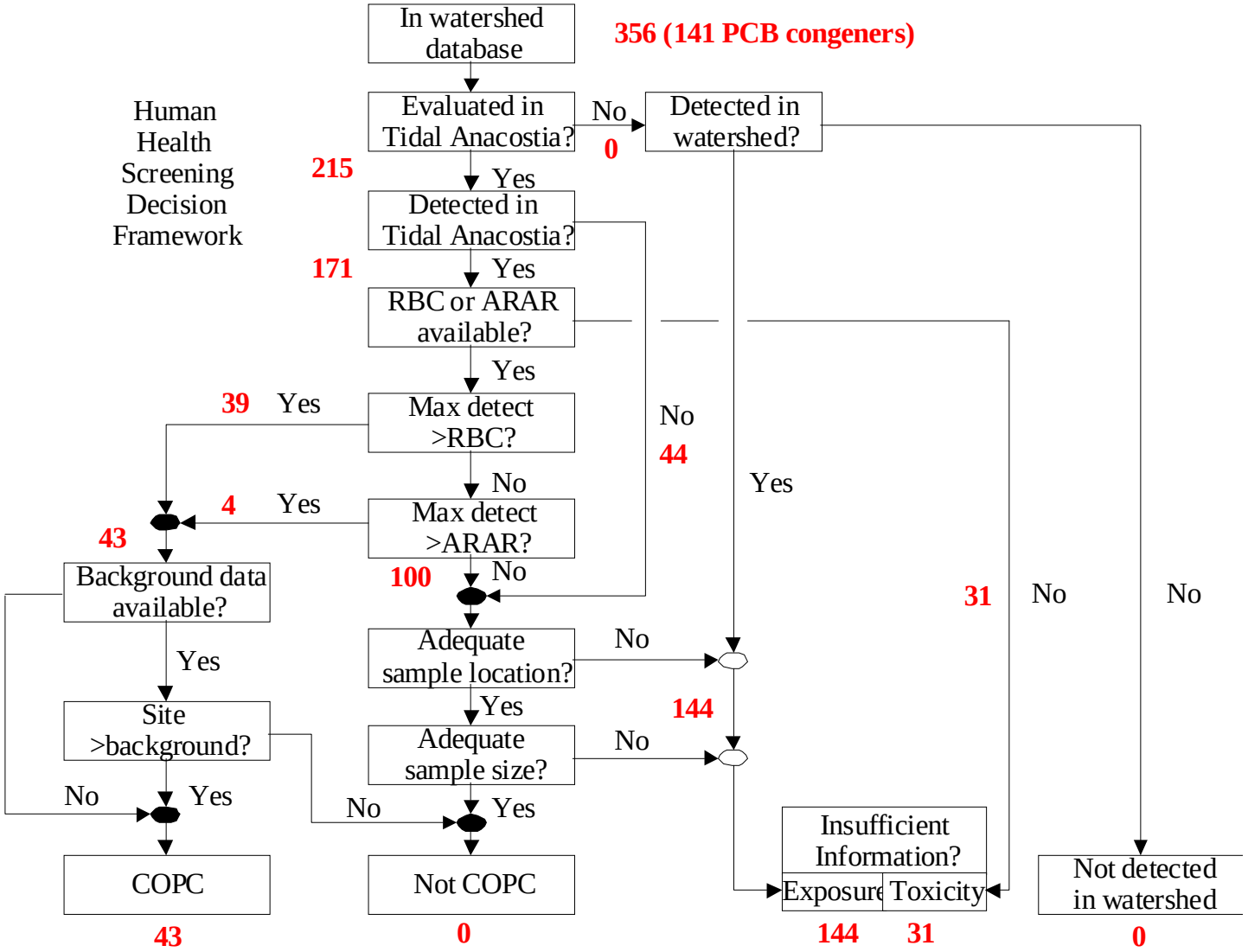


Figure 6-3. Decision Framework for Human Health Risk Screening. The numbers of chemicals that were sorted at each major decision point are shown.

Table 6-1. Exposure Factors Used in Soil RBCs (Industrial Scenario)

Exposure Variable	Units	Value	Symbol
Target cancer risk	probability	10^{-6}	TR
Target hazard quotient	unitless	0.1	THQ
Carcinogenic potency slope oral	risk/mg/kg/day	chemical-dependent	CPS_o
Reference dose oral	mg/kg/day	chemical-dependent	RfD_o
Body weight, adult	kg	70	BW_a
Averaging time (carcinogens)	days	25,550	AT_c
Averaging time (non-carcinogens)	days	ED x 365	AT_n
Soil ingestion, adult	mg/day	100	IRS_a
Exposure frequency, occupational	days/year	250	EF_o
Exposure duration	year	25	ED_o
Fraction of contaminated soil ingested	unitless	0.5	FC

From U.S. EPA, 1999

Table 6-2. Exposure Factors Used in Tap Water RBCs

Exposure Variable	Units	Value	Symbol
Target cancer risk	probability	10^{-6}	TR
Target hazard quotient	unitless	0.1	THQ
Carcinogenic potency slope oral	risk/mg/kg/day	chemical-dependent	CPS_o
Carcinogenic potency slope inhaled	risk/mg/kg/day	chemical-dependent	CPS_i
Reference dose oral	mg/kg/day	chemical-dependent	RfD_o
Reference dose inhaled	mg/kg/day	chemical-dependent	RfD_i
Body weight, adult	kg	70	BW_a
Averaging time (carcinogens)	days	25,550	AT_c

Averaging time (non-carcinogens)	days	ED x 365	AT_n
Volatilization factor	L/m^3	0.5	K
Inhalation factor, age-adjusted	m^3-year/kg-day	11.66	IFA_{adj}
Tap water ingestion factor, age-adjusted	L-year/kg-day	1.09	IFW_{adj}
Inhalation rate, adult	m^3/day	20	IRA_a
Tap water ingestion rate, adult	L/day	2	IRW_a
Exposure frequency, residential	days/year	350	EF_r
Exposure duration, total	year	30	ED_{tot}

From U.S. EPA, 1999

Table 6-3. Exposure Factors Used in Fish Tissue RBCs

Exposure Variable	**Units**	**Value**	**Symbol**
Target cancer risk	probability	10^{-6}	TR
Target hazard quotient	unitless	0.1	THQ
Carcinogenic potency slope oral	risk/mg/kg/day	chemical-dependent	CPS_o
Reference dose oral	mg/kg/day	chemical-dependent	RfD_o
Body weight, adult	kg	70	BW_a
Averaging time (carcinogens)	days	25,550	AT_c
Averaging time (non-carcinogens)	days	ED x 365	AT_n
Fish ingestion rate	g/day	54	IRF
Exposure frequency, residential	days/year	350	EF_r
Exposure duration, total	year	30	ED_{tot}

From U.S. EPA, 1999

Table 6-4. Parameter Values Used in the U.S. Interim Adult Lead Methodology " \f F

Parameter	Units	Value	Symbol
Goal for the 95th percentile blood lead concentration among fetuses	Φg/dL	10	$PbB_{fetal.\ 0.95,\ goal}$
Individual geometric standard deviation of blood lead concentration	unitless	1.8	$GSD_{i,adult}$
Constant of proportionality between fetal and maternal blood lead concentration at birth	unitless	0.9	$R_{fetal,maternal}$
Typical blood lead concentration in adults in absence of exposure to the site that is being assessed	Φg/dL	2.0	$PbB_{adult,0}$
Biokinetic slope factor relating increase in typical adult blood lead level to average daily lead uptake	Φg/dL per Φg/day	0.4	BKSF
Ingestion rate of soil	g/day	0.05	IR_s
Exposure frequency	days/year	219	EF_s
Absolute gastrointestinal absorption fraction for ingested lead in soil	unitless	0.12	AF_s
Averaging time	days/year	365	AT
Parameter values yield a soil RBC of 800 ppm (From U.S. EPA, 1996)			

Table 6-5. Toxicity Equivalency Factors (TEFs) for *Dioxin-like* Congeners of Chlorinated Dibenzo-p-dioxins (CDDs) and Chlorinated Dibenzofurans (CDFs)

CAS No.	*Congener or Homolog*	*CHEMCODE[1]*	*TEF*	
			EPA2	*WHO3*
40321764	*PCDD12378*	*PCD12378*	*0.5*	*1.0*
57117416	*PCDF12378*	*PCF12378*	*0.05*	*0.05*
57117314	*PCDF23478*	*PCF23478*	*0.5*	*0.5*
1746016	*TCDD2378 (dioxin)*	*PCD2378*	*1.0*	*1.0*
51207319	*TCDF2378*	*PCF2378*	*0.1*	*0.1*
39227286	*H6CDD123478*	*PCD123478*	*0.1*	*0.1*

57653857	*H6CDD123678*	*PCD123678*	*0.1*	*0.1*
19408743	*H6CDD123789*	*PCD123789*	*0.1*	*0.1*
70648269	*H6CDF123478*	*PCF123478*	*0.1*	*0.1*
57117449	*H6CDF123678*	*PCF123678*	*0.1*	*0.1*
72918219	*H6CDF123789*	*PCF123789*	*0.1*	*0.1*
60851345	*H6CDF234678*	*PCF234678*	*0.1*	*0.1*
35822469	*H7CDD1234678*	*PCD1234678*	*0.01*	*0.01*
67562394	*H7CDF1234678*	*PCF1234678*	*0.01*	*0.01*
55673897	*H7CDF1234789*	*PCF1234789*	*0.01*	*0.01*
39001020	*Octachlorodibenzofuran*	*OCDF*	*0.001*	*0.0001*
3268879	*Octachlorodibenzo-p-dioxin*	*OCDD*	*0.001*	*0.0001*

[1]Identifying code used in screening database and NOAA (2000)
[2]U.S. EPA, 1989
[3]Van den Berg et al., 1998

6.2.4 BACKGROUND SCREEN

When site-specific background chemical concentrations are available, the concentrations of chemicals on the preliminary list of COPCs would be compared against background to determine whether the concentration is higher than background; chemicals that are not demonstrably higher than background may then be placed in the not-COPC group. No site-specific background concentrations were identified for tidal Anacostia River media for use in the screening human health risk assessment. In the absence of background data, all chemicals for which the maximum concentration exceeded an RBC or an ARAR were considered to be COPCs.

6.2.5 CATEGORY 1 CHEMICALS - CHEMICALS OF POTENTIAL CONCERN (COPCS)

COPCs include those chemicals that were: 1) detected in tidal Anacostia River sediments, surface water or fish; and 2) for which the maximum concentration in any single medium exceeded an RBC or ARAR. Based on these screening criteria, 43 chemicals were categorized as COPCs (Table 6-6 a); 39 chemicals are COPCs in fish, 7 in river sediment and 5 in river water; six of the chemicals are COPCs for more than one media.

Fish tissue COPCs fall into several chemical classes. Seventeen are chlorinated dibenzodioxins or dibenzofurans; 12 are organic pesticides: aldrin, (-HCH (lindane), HCB, DDT, DDE, dieldrin, chlordane (or transformation products), or heptachlor; 2 are PCBs, including Aroclor 1260; and 4 are metals: lead, mercury, arsenic and cadmium. The remaining fish tissue COPCs are ∀-HCH, heptachlor epoxide, bis(2-ethylhexyl)phthalate and di-N-octyl phthalate. Four of the COPCs for fish were placed in Category 1 for exceeding an ARAR: transchlordane, cischlordane, gammachlordane and lead.

The 7 COPCs in sediment include two chemicals (or chemical mixtures) that are also COPCs in water and fish tissue: arsenic and total PCBs and one chemical that is also a COPC in fish tissue: Aroclor 1260. The remaining 4 sediment COPCs include the following PAHs: benzo(a)pyrene, dibenz(a,h)anthracene, benz(a)anthracene and benzo(b)fluoranthene.

The 5 COPCs in water include two chemicals (or chemical mixtures) that are also COPCs in fish tissue and sediment: total PCBs and arsenic, and three that are also COPCs in fish tissue: heptachlor (pesticide), DDE and DDT.

Table 6-6b presents the COPCs for each environmental medium, sorted by the ratio of the maximum detected concentration to the RBC (max/RBC). The max/RBC ratio provides the magnitude by which the maximum concentration exceeds the RBC. A high max/RBC ratio would indicate a greater potential for concern that a given chemical may pose a risk at reasonable maximal exposure (RME), given the conservative assumptions in the screening assessment. The max/RBC ratio range was 1.1–2911 for fish tissue and 1.2–34 for sediment. In fish tissue, total PCBs had the highest max/RBC ratio; in sediment, benzo(a)pyrene had the highest ratio. The max/RBC ratio range was 1.1–380 for water, total PCBs had the highest max/RBC ratio. The three sediment COPCs that are also COPCs in fish tissue, total PCBs, Aroclor 1260, and arsenic represent the first, second and fifth highest max/RBC in fish tissue, respectively, and all 3 had max/RBC ratios in fish tissue that exceeded 100.

Figure 6-4 shows the geographic distribution of the maximum concentrations for COPCs, Table 6-6c provides the study identification numbers, sample locations and the sampling dates corresponding to the COPCs. Sampling stations 16330 (downstream from the confluence of the Northwest and Northeast Branches), 3 (near and upstream from Kingman Lake), and stations 4 and LA (nearly co-located with station 4, near and upstream from the South Capital St Bridge) are the most widely separated stations at which fish contaminants were measured. The considerable commonality of the COPCs and their respective chemical classes detected at these stations would suggest either their relatively widespread

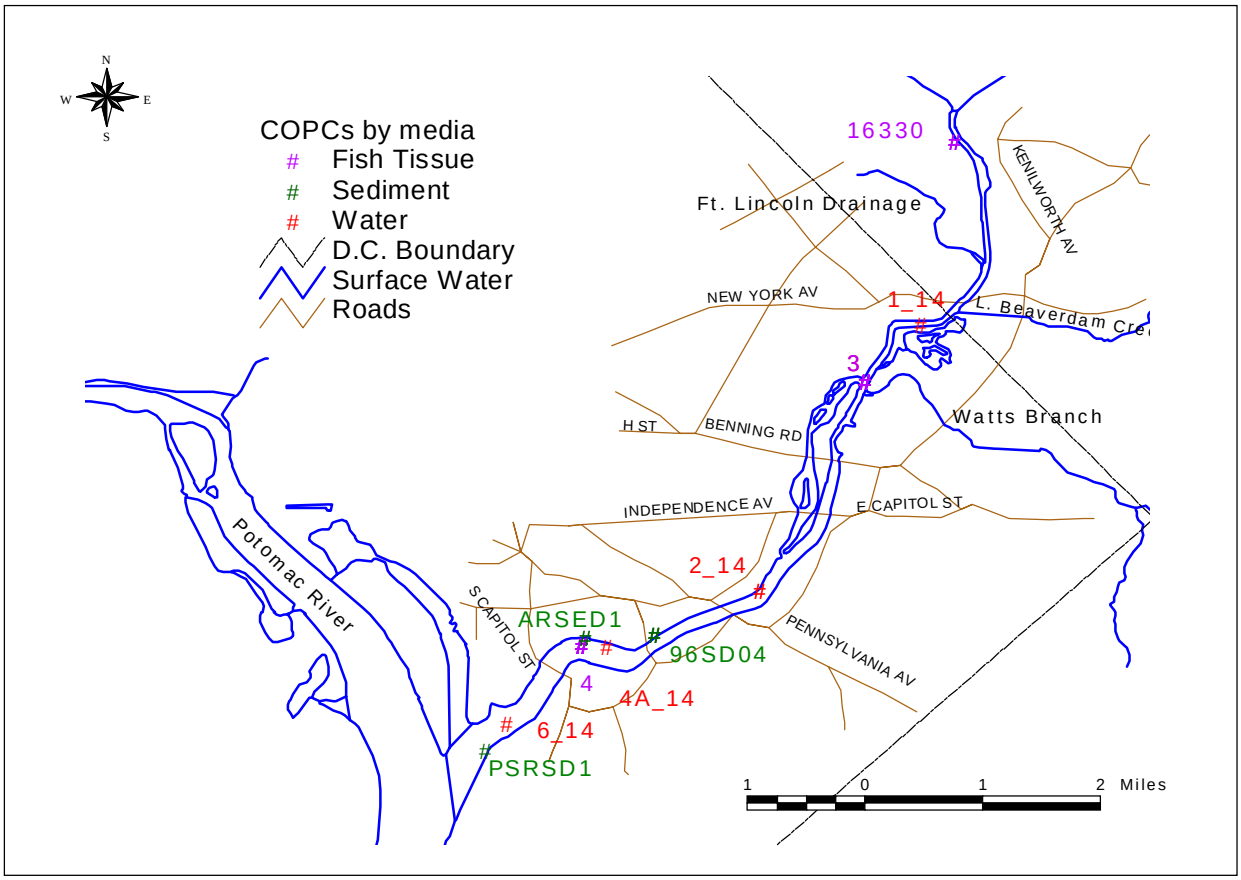


Figure 6-4. Sample Stations Where the Maximum Concentrations of the Chemicals of Potential Concern (COPCs) were Detected. Table 6-6c provides the Study ID numbers and the sample dates corresponding to the COPCs.

occurrence in the tidal Anacostia or the ranging patterns of the fish species sampled. COPCs detected at all of these regions of the tidal river include the pesticides chlordane, heptachlor, and lindane; chlorinated dibenzodioxins and dibenzofurans; and polychlorinated biphenyls. Note that co-location of COPCs does not necessarily indicate concurrence, as samples were collected over a time-span of 5 years.

Hazard identification summaries for each COPC are presented in Section 6.2.10

6.2.6 CATEGORY 2 CHEMICALS - NOT CHEMICALS OF POTENTIAL CONCERN (NOT COPCS)

Although 104 chemicals had maximum concentrations that did not exceed an RBC, none of these chemicals qualified for inclusion in the Not COPC category. This outcome was primarily the result of either low sample numbers or inadequate geographic distribution of samples, either of which resulted in their inclusion in Category 3 (Insufficient Information Related to Exposure). Four of the 104 chemicals exceeded an ARAR and were placed in Category 1. In addition, 44 chemicals that were not detected in any sample were also placed in Category 3 due to inadequate sample number or inadequate geographic distribution of samples.

6.2.7 CATEGORY 3 CHEMICALS - INSUFFICIENT INFORMATION RELATED TO EXPOSURE

The screening of chemicals against criteria for sample number and geographic distribution (see Section 6.2.1 for criteria) resulted in the inclusion in Category 3 of all 100 detected chemicals that were not categorized as COPCs (Category 1) (Table 6-7a) and the 44 undetected chemicals (Table 6-7b). The primary basis for this outcome is as follows: 1) there are no records in the database of sediment samples for sections of the river channel upstream of Hickey Run; 2) relatively few sediment samples are recorded for the section of the river between Benning Road Bridge and the Pennsylvania Avenue Bridge, none of which were analyzed for Aroclors; and 3) there is only one water sampling station north of the Independence Avenue Bridge. As a result, large sections of the river are not represented in the data on chemical concentrations in sediment and in the water column. These include areas that potentially may have received chemical inputs from the Northeast and Northwest Branches, the Fort Lincoln drainage area, the Kenilworth Marsh, Kingman Lake and other urban drainage sources along these sections of the river. In addition, the database does not contain any information on the concentration of dioxins, furans or PAHs in the water column.

Category 3 includes numerous chemicals that are members of chemical classes represented in the COPC list, including PCBs (Aroclors), several PAHs, a variety of pesticides, several metals. Thus, it would not be particularly surprising if some of these chemicals were to be reclassified as COPCs, based on the results of additional sampling of the river or the inclusion of additional, existing sample information in the database.

The lack of data from the upper tidal Anacostia River is a significant data gap. Several studies have identified dissolved and/or particulate influx from the Northeast and Northwest Branches as potentially significant contributors to chemical loadings to the tidal Anacostia River. In addition, sedimentation rates in the upper tidal river, would suggest that particulate chemical loadings from the upper tributaries may deposit in the upper tidal river. Surface water movement and particulate transport are not sufficiently characterized in the upper tidal Anacostia River to determine whether particulate influx from the upper tributaries typically deposit in, or pass through, the upper tidal river area. The available information is suggestive that concentrations in surface water, sediment, and/or biota in the upper tidal river may be high enough for certain chemicals that are not yet identified as COPCs to be identified as COPCs if further data were available. The uncertainty regarding chemical concentrations in media in the upper tidal river may be resolved with acquisition of additional data.

The screening assessment also determined if there were any chemicals that were detected in other areas of

the watershed but were not evaluated in the tidal Anacostia River. These would have been included in Category 3. No chemicals qualified under this criteria.

6.2.8 CATEGORY 4 - INSUFFICIENT INFORMATION RELATED TO TOXICITY

Thirty-one chemicals could not be evaluated against toxicity criteria because of the lack of an RBC (Table 6-8); however, three of the chemicals were placed in Category 1 for exceeding ARARs: cischlordane, gammachlordane and transchlordane. Twelve of the Category 4 chemicals are PAHs which, as a chemical class, are represented on the COPC list. Twelve Category 4 chemicals are pesticides or structural analogs, including several structural or compositional analogs of the following chemicals that are COPCs: BHCs, hexachlorocyclohexane-delta (lindane), the ortho-para isomers of DDD, DDE and DDT, oxychlordane and transchlordane. Several other chemicals in Category 4 are structurally and/or toxicologically similar to chemicals that are identified as COPCs. Arsenic III is listed as a Category 4 chemical even though the maximum concentration of arsenic III reported in water column samples (1.8E–01 ppm) exceeded the cancer-based RBC for inorganic arsenic (4.46E–02 ppm). Monomethyl and dimethyl arsenic are also included in the Category 4 list.

6.2.9 CATEGORY 5 - NOT EVALUATED IN TIDAL ANACOSTIA RIVER AND NOT DETECTED IN WATERSHED

There were no chemicals in this category.

6.2.10 HAZARD IDENTIFICATION FOR COPCS

Hazard identification information is summarized in Appendix B for each COPC identified in the screening assessment (Category 1). The summaries include the hazard basis for the RBC, cancer or non-cancer effects, and the relevant dose-response information available from U.S. EPA (2000).

6.2.11 UNCERTAINTY ASSESSMENT FOR SCREENING RESULTS

The methodology used in this screening assessment is intended to identify those chemicals in the tidal Anacostia that might be of potential concern as human health risks (COPCs) so that additional data can be collected on these chemicals that would support quantitative estimates of risk. In order to ensure that all COPCs are identified and that no chemicals are misclassified as *Not COPCs*, the risk based screening approach is intentionally designed to have a health protective bias. This bias or conservatism derives from the following:

X use of the maximum concentration as the exposure concentration term in the screening assessment;

X use of risk criteria (e.g., cancer risk of 10^{-6} and hazard quotient of 0.1) that are generally considered acceptable for environmental exposures to chemicals;

X use of exposure factors that represent a reasonable maximum exposure (RME) estimate for ingestion of soil, which probably overestimates the RME estimate for river sediment; and

X use of exposure factors for exposure to residential tap water, which would be expected to substantially overestimate RME estimates for ingestion of surface water because river water is not used for tap water in the region.

The above notwithstanding, important uncertainties attend identification of COPCs based on this methodology, given the available data. The uncertainties were considered too large to support a definitive

classification of any chemicals into the *Not COPC* category. As a result, a large number of chemicals were assigned to other categories based on the major source of uncertainty: 1) those related to representativeness of the samples used to estimate a maximum concentration (Category 3) and; 2) those related to the lack of toxicity values to support RBCs for use in the screening assessment (Category 4).

Uncertainties in Representativeness of the Samples

Data from fish, sediment and water samples that are included in NOAA 2000 database were used to estimate the maximum exposure concentrations that would occur in human receptors that contact the river. However, limitations in the geographic and temporal distribution of the samples included in the NOAA 2000 database made such estimates highly uncertain for most of the chemicals that entered the screening assessment. For example, there are no sediment samples for sections of the river channel upstream of Hickey Run and relatively few sediment samples between Benning Road Bridge and the Pennsylvania Avenue Bridge, none of which were analyzed for Aroclors. Furthermore, there is only one water column sampling location upstream of the Independence Avenue Bridge and no stations upstream from Lower Beaverdam Creek. There is no information on the concentration of dioxins, furans, or PAHs in the water column. Existing data were collected at various times and do not represent a random sample of the river either spatially or temporally. As a result of these limitations, there are very little or no data for large sections of the river, including potentially important inputs such as the Northeast and Northwest Branches, Fort Lincoln drainage area, Kenilworth Marsh, Kingman Lake and other urban drainage sources. Therefore, it is possible that some of the chemicals that were not classified as COPCs (e.g., Category 3, Table 6-7a,b) may actually have maximum concentrations that exceed RBCs or ARARs in areas that were not sampled. Also, because of the dynamic nature of the river flow in response to storms and annual weather patterns, the maximum concentrations obtained from the available data may not reflect current or future maximum values.

In order to account for these uncertainties in the screening assessment, the adequacy of sampling was evaluated in terms of both the number of samples and geographic distribution of sampling within the tidal Anacostia River. Chemicals that did not satisfy minimum criteria for number and geographic distribution of samples were placed into Category 3 (Table 6-7a,b). The criteria were a minimum of three samples representing the upper, middle and lower regions of the tidal Anacostia River, including areas immediately downstream from the major inflows and catchments: 1) Northeast and Northwest Branches; 2) Fort Lincoln Drainage; 3) Lower Beaver Dam Creek; 4) Hickey Run and Watts Branch; 5) Washington Channel area; 6) Kenilworth Marsh; and 7) Kingman Lake.

The eight studies that contributed information to the NOAA 2000 data base collected sediment samples from depth intervals ranging from 0–3 to 0–30.48 cm. Ideally, data used in the screening risk assessment should represent the concentrations of chemicals in media to which receptors may come into contact. Given the dynamic erosion/deposition patterns of a river system, it is likely that the appropriate depth for collecting sediment samples would vary depending upon the location from which samples were collected. However, due to the 'smoothing' effect of averaging concentrations over increasing volumes of media, it is possible that the maximum concentration of a chemical detected in sediment at a particular location may be less than the maximum that would have been detected if a smaller interval of the sediment had been sampled.

Uncertainties Related to Lack of Toxicity Information

Risk-based Concentrations (RBCs) were not available for 29 chemicals that were detected in the tidal Anacostia River (Table 6-8). Several of these chemicals are structurally and/or toxicologically similar to chemicals that are identified as COPCs. These include the ortho-para isomers of DDD, DDE and DDT; arsenic III and mono- and dimethyl arsenic; oxychlordane and transchlordane; hexachlorocyclohexane delta and total BHC, and several PAHs. Had RBCs been available for these chemicals, it is possible that

some would have had maximum concentrations that exceeded their respective RBCs.

Table 6-6a. Chemicals of Potential Concern (COPCs) Identified in the Human Health Screening Assessment (sorted by medium and chemical name)

Media	CAS No.	Chemical Name	Det Freq	Max	Units	RBC / ARAR[1]	RBC Basis[2]	Max Location[3]	Max/ RBC
Fish Tissue	309002	Aldrin	8 / 32	2.31E-03	ppm	1.86E-04	C	LA	12
Fish Tissue	11096825	Aroclor 1260	3 / 3	4.50E-01	ppm	1.58E-03	C	16330	285
Fish Tissue	7440382	Arsenic	10 / 16	2.66E-01	ppm	2.10E-03	C	LA	127
Fish Tissue	117817	Bis(2ethylhexyl) phthalate	16 / 18	6.40E-01	ppm	2.25E-01	C	3	2.8
Fish Tissue	7440439	Cadmium	15 / 16	2.00E-01	ppm	1.35E-01	N	16330	1.5
Fish Tissue	319846	Hexachlorocyclohexanealpha	13 / 32	8.00E-03	ppm	5.01E-04	C	16330	16
Fish Tissue	58899	Hexachlorocyclohexanegamma (Lindane)	12 / 32	2.58E-03	ppm	2.43E-03	C	LA	1.1
Fish Tissue	118741	Hexachlorobenzene (HCB)	13 / 32	4.98E-03	ppm	1.97E-03	C	LA	2.5
Fish Tissue	60571	Dieldrin	41 / 44	5.20E-02	ppm	1.97E-04	C	LA	264
Fish Tissue	1024573	Heptachlor epoxide	26 / 32	1.70E-02	ppm	3.47E-04	C	4	49
Fish Tissue	76448	Heptachlor (pesticide)	12 / 32	6.10E-03	ppm	7.01E-04	C	3	8.7
Fish Tissue	117840	DiNoctyl phthalate	16 / 18	6.70E+00	ppm	2.70E+00	N	4	2.5
Fish Tissue	3268879	Octachlorodibenzopdioxin	18 / 18	5.71E-05	pm	2.10E-05	C	4	2.7
Fish Tissue	39001020	Octachlorodibenzofuran	18 / 18	9.22E-05	ppm	2.10E-05	C	3	4.4
Fish Tissue	1336363	PCB, total	43 / 44	4.60E+00	ppm	1.58E-03	C	LA	2911
Fish Tissue	35822469	H7CDD1234678	15 / 18	6.20E-06	ppm	2.10E-06	C	4	3.0
Fish Tissue	39227286	H6CDD123478	16 / 18	5.70E-06	ppm	2.10E-07	C	3	27
Fish Tissue	57653857	H6CDD123678	16 / 18	7.40E-06	ppm	2.10E-07	C	3	35
Fish Tissue	40321764	PCDD12378	16 / 18	3.90E-06	ppm	4.20E-07	C	4	9.3
Fish Tissue	19408743	H6CDD123789	17 / 18	1.03E-05	ppm	2.09E-07	C	3	49
Fish Tissue	1746016	TCDD2378 (dioxin)	18 / 18	2.80E-06	ppm	2.10E-08	C	3	133
Fish Tissue	67562394	H7CDF1234678	18 / 18	1.96E-05	ppm	2.10E-06	C	3	9.3
Fish Tissue	70648269	H6CDF123478	18 / 18	1.00E-05	ppm	2.10E-07	C	3	48
Fish Tissue	55673897	H7CDF1234789	18 / 18	2.55E-06	ppm	2.10E-06	C	4	1.2
Fish Tissue	57117449	H6CDF123678	18 / 18	8.10E-06	ppm	2.10E-07	C	3	39
Fish Tissue	57117416	PCDF12378	17 / 18	5.00E-06	ppm	4.20E-06	C	3	1.2
Fish Tissue	72918219	H6CDF123789	16 / 18	9.50E-06	ppm	2.10E-07	C	3	45

Table 6-6a. Chemicals of Potential Concern (COPCs) Identified in the Human Health Screening Assessment (sorted by medium and chemical name)

Media	CAS No.	Chemical Name	Det Freq	Max	Units	RBC / ARAR[1]	RBC Basis[2]	Max Location[3]	Max/ RBC
Fish Tissue	60851345	H6CDF234678	17 / 18	5.00E-06	ppm	2.10E-07	C	3	24
Fish Tissue	57117314	PCDF23478	18 / 18	4.75E-06	ppm	4.20E-07	C	3	11
Fish Tissue	51207319	TCDF2378	18 / 18	4.80E-06	ppm	2.10E-07	C	3	23
Fish Tissue	72548	p,p'DDD	42 / 44	4.80E-01	ppm	1.31E-02	C	LA	3
Fish Tissue	72559	p,p'DDE	43 / 44	5.00E-01	ppm	9.28E-03	C	LA	54
Fish Tissue	50293	p,p'DDT	30 / 44	5.10E-02	ppm	9.28E-03	C	LA	5.5
Fish Tissue	57749	Total chlordane	13 / 15	8.00E-01	ppm	9.01E-03	C	LA	89
Fish Tissue[4]	5103719	Cischlordane	29 / 29	3.40E-01	ppm	8.30E-03		4	41
Fish Tissue[4]	5566347	Gammachlordane	29 / 29	9.00E-02	ppm	8.30E-03		3	11
Fish Tissue[4]	7439921	Lead	16 / 16	4.20E+00	ppm	1.30E+00		16330	3.2
Fish Tissue	7439976	Mercury	16 / 16	1.59E-01	ppm	1.40E-02	N	16330	11
Fish Tissue[4]	5103742	Transchlordane	10 / 12	2.30E-01	ppm	8.30E-03	N	LA	28
Sediment	11096825	Aroclor 1260	15 / 25	1.20E+01	ppm	2.86E+00	C	ARSED1	4.2
Sediment	7440382	Arsenic	33 / 34	2.69E+01	ppm	3.82E+00	C	PSRSD1	7.0
Sediment	56553	Benz(a)anthracene	44 / 45	1.60E+01	ppm	7.84E+00	C	96SD04	2.0
Sediment	53703	Dibenz(a,h)anthracene	37 / 45	6.90E+00	ppm	7.84E-01	C	96SD04	8.8
Sediment	50328	Benzo(a)pyrene	44 / 45	2.70E+01	ppm	7.84E-01	C	96SD04	34
Sediment	205992	Benzo(b)fluoranthene	41 / 44	9.20E+00	ppm	7.84E+00	C	96SD04	1.2
Sediment	1336363	PCB, total	33 / 45	1.20E+01	ppm	2.86E+00	C	ARSED1	4.2
Water	7440382	Arsenic	33 / 33	6.60E-01	ppb	4.46E-02	C	1-14	15
Water[4]	76448	Heptachlor (pesticide)	9 / 38	2.85E-04	ppb	2.1E-04		4A_14	1.4
Water[4]	1336363	PCBs (total)	38 / 38	1.72E-02	ppb	4.5E-05		2_14	380
Water[4]	72559	p,p'-DDE	38 / 38	1.45E-03	ppb	5.9E-04		2_14	2.5
Water[4]	50293	p,p'-DDT	27 / 38	6.49E-04	ppb	5.9E-04		6_14	1.1

Table 6-6a. Chemicals of Potential Concern (COPCs) Identified in the Human Health Screening Assessment (sorted by medium and chemical name)

Media	CAS No.	Chemical Name	Det Freq	Max	Units	RBC / ARAR[1]	RBC Basis[2]	Max Location[3]	Max/ RBC

Det Freq, detection frequency (number of detects/number to samples); Max, maximum concentration; RBC, risk-based concentration
[1](a)E(b) refers to [a≅10[b]]
[2] C, cancer; N, non-cancer
[3]Refers to location codes in NOAA (2000)
[4]Chemicals that were placed in Category 1 for exceeding an ARAR

Table 6-6b Chemicals of Potential Concern (COPCs) Identified in the Human Health Screening Assessment (sorted by medium and Max/RBC)

Media	CAS No.	Chemical Name	Det Freq	Max	Units	RBC / ARAR[1]	RBC Basis2	Max Location3	Max/ RBC
Fish Tissue	1336363	PCB, total	43 / 44	4.60E+00	ppm	1.58E-03	C	LA	2911
Fish Tissue	11096825	Aroclor 1260	3 / 3	4.50E-01	ppm	1.58E-03	C	16330	285
Fish Tissue	60571	Dieldrin	41 / 44	5.20E-02	ppm	1.97E-04	C	LA	264
Fish Tissue	1746016	TCDD2378 (dioxin)	18 / 18	2.80E-06	ppm	2.10E-08	C	3	133
Fish Tissue	7440382	Arsenic	10 / 16	2.66E-01	ppm	2.10E-03	C	LA	127
Fish Tissue	57749	Total chlordane	13 / 15	8.00E-01	ppm	9.01E-03	C	LA	89
Fish Tissue	72559	p,p'DDE	43 / 44	5.00E-01	ppm	9.28E-03	C	LA	54
Fish Tissue	19408743	H6CDD123789	17 / 18	1.03E-05	ppm	2.09E-07	C	3	49
Fish Tissue	1024573	Heptachlor epoxide	26 / 32	1.70E-02	ppm	3.47E-04	C	4	49
Fish Tissue	70648269	H6CDF123478	18 / 18	1.00E-05	ppm	2.10E-07	C	3	48
Fish Tissue	72918219	H6CDF123789	16 / 18	9.50E-06	ppm	2.10E-07	C	3	45
Fish Tissue4	5103719	Cischlordane	29 / 29	3.40E-01	ppm	8.30E-03		4	41
Fish Tissue	57117449	H6CDF123678	18 / 18	8.10E-06	ppm	2.10E-07	C	3	39
Fish Tissue	72548	p,p'DDD	42 / 44	4.80E-01	pm	1.31E-02	C	LA	37
Fish Tissue	57653857	H6CDD123678	16 / 18	7.40E-06	ppm	2.10E-07	C	3	35
Fish Tissue[4]	5103742	Transchlordane	10 / 12	2.30E-01	ppm	8.30E-03	N	LA	28
Fish Tissue	39227286	H6CDD123478	16 / 18	5.70E-06	ppm	2.10E-07	C	3	27

Table 6-6b Chemicals of Potential Concern (COPCs) Identified in the Human Health Screening Assessment (sorted by medium and Max/RBC)

Media	CAS No.	Chemical Name	Det Freq	Max	Units	RBC / ARAR[1]	RBC Basis2	Max Location3	Max/ RBC
Fish Tissue	60851345	H6CDF234678	17 / 18	5.00E-06	ppm	2.10E-07	C	3	24
Fish Tissue	51207319	TCDF2378	18 / 18	4.80E-06	ppm	2.10E-07	C	3	23
Fish Tissue	319846	Hexachlorocyclohexanealpha	13 / 32	8.00E-03	ppm	5.01E-04	C	16330	16
Fish Tissue	309002	Aldrin	8 / 32	2.31E-03	ppm	1.86E-04	C	LA	12
Fish Tissue[4]	5566347	Gammachlordane	29 / 29	9.00E-02	ppm	8.30E-03		3	11
Fish Tissue	7439976	Mercury	16 / 16	1.59E-01	ppm	1.40E-02	N	16330	11
Fish Tissue	57117314	PCDF23478	18 / 18	4.75E-06	ppm	4.20E-07	C	3	11
Fish Tissue	67562394	H7CDF1234678	18 / 18	1.96E-05	ppm	2.10E-06	C	3	9.3
Fish Tissue	40321764	PCDD12378	16 / 18	3.90E-06	ppm	4.20E-07	C	4	9.3
Fish Tissue	76448	Heptachlor (pesticide)	12 / 32	6.10E-03	ppm	7.01E-04	C	3	8.7
Fish Tissue	50293	p,p'DDT	30 / 44	5.10E-02	ppm	9.28E-03	C	LA	5.5
Fish Tissue	39001020	Octachlorodibenzofuran	18 / 18	9.22E-05	ppm	2.10E-05	C	3	4.4
Fish Tissue[4]	7439921	Lead	16 / 16	4.20E+00	ppm	1.30E+00		16330	3.2
Fish Tissue	35822469	H7CDD1234678	15 / 18	6.20E-06	ppm	2.10E-06	C	4	3.0
Fish Tissue	117817	Bis(2ethylhexyl) phthalate	16 / 18	6.40E-01	ppm	2.25E-01	C	3	2.8
Fish Tissue	3268879	Octachlorodibenzopdioxin	18 / 18	5.71E-05	ppm	2.10E-05	C	4	2.7
Fish Tissue	118741	Hexachlorobenzene (HCB)	13 / 32	4.98E-03	ppm	1.97E-03	C	LA	2.5
Fish Tissue	117840	DiNoctyl phthalate	16 / 18	6.70E+00	ppm	2.70E+00	N	4	2.5
Fish Tissue	7440439	Cadmium	15 / 16	2.00E-01	ppm	1.35E-01	N	16330	1.5
Fish Tissue	57117416	PCDF12378	17 / 18	5.00E-06	ppm	4.20E-06	C	3	1.2
Fish Tissue	55673897	H7CDF1234789	18 / 18	2.55E-06	ppm	2.10E-06	C	4	1.2
Fish Tissue	58899	Hexachlorocyclohexanegamma (Lindane)	12 / 32	2.58E-03	ppm	2.43E-03	C	LA	1.1
Sediment	50328	Benzo(a)pyrene	44 / 45	2.70E+01	ppm	7.84E-01	C	96SD04	34
Sediment	53703	Dibenz(a,h)anthracene	37 / 45	6.90E+00	ppm	7.84E-01	C	96SD04	8.8
Sediment	7440382	Arsenic	33 / 34	2.69E+01	ppm	3.82E+00	C	PSRSD1	7.0
Sediment	11096825	Aroclor 1260	15 / 25	1.20E+01	ppm	2.86E+00	C	ARSED1	4.2
Sediment	1336363	PCBS, total	33 / 45	1.20E+01	ppm	2.86E+00	C	ARSED1	4.2

Table 6-6b Chemicals of Potential Concern (COPCs) Identified in the Human Health Screening Assessment (sorted by medium and Max/RBC)

Media	CAS No.	Chemical Name	Det Freq	Max	Units	RBC / ARAR[1]	RBC Basis2	Max Location3	Max/ RBC
Sediment	56553	Benz(a)anthracene	44 / 45	1.60E+01	ppm	7.84E+00	C	96SD04	2.0
Sediment	205992	Benzo(b)fluoranthene	41 / 44	9.20E+00	ppm	7.84E+00	C	96SD04	1.2
Water[4]	1336363	PCBs (total)	38 / 38	1.72E-02	ppb	4.5E-05		2_14	380
Water	7440382	Arsenic	33 / 33	6.60E-01	ppb	4.46E-02	C	1_14	15
Water[4]	72559	p,p'-DDE	38 / 38	1.45E-03	ppb	5.9E-04		2_14	2.5
Water[4]	76448	Heptachlor (pesticide)	9 / 38	2.85E-04	ppb	2.1E-04		4A_14	1.4
Water[4]	50293	p,p'-DDT	27 / 38	6.49E-04	ppb	5.9E-04		6_14	1.1

Det Freq, detection frequency (number of detects/number to samples); Max, maximum concentration; RBC, risk-based concentration

[1](a)E(b) refers to [a≅10[b]]

[2] C, cancer; N, non-cancer

[3]Refers to location codes in NOAA (2000)

[4]Chemicals that were placed in Category 1 for exceeding an ARAR

Table 6-6c. Category 1 - COPCs (grouped by medium and sampling station)

Media	Sample Station	COPCs	Sampling Date / Study ID
Fish Tissue	16330	Aroclor 1260, cadmium, chlordane, Hexachlorocyclohexanealpha, lead, mercury	Unknown / Study ID #A1
	3	Bis(2ethylhexyl) phthalate, Gammachlordane, Heptachlor (pesticide), Octachlorodibenzofuran, H6CDD123478, H6CDD123678, H6CDD123789, TCDD2378 (dioxin), H7CDF1234678, H6CDF123478, H6CDF123678, PCDF12378, H6CDF123789, H6CDF234678, PCDF23478, TCDF2378	1989-1992 / Study ID #4
	4	Alphachlordane, Heptachlor epoxide, DiNoctyl phthalate, Octachlorodibenzopdioxin, H7CDD1234678, PCDD12378, H7CDF1234789	1989-1992 / Study ID #4
	LA[1]	Aldrin, Arsenic, Hexachlorocyclohexanegamma (Lindane), Hexachlorobenzene (HCB), Dieldrin, PCBS (total), p,p'DDD, p,p'DDE, p,p'DDT, Total chlordane, (alpha+cis+oxy+trans), Transchlordane	August 1987 / Study ID #5
Sediment	96SD04	Benz(a)anthracene, Dibenz(a,h)anthracene, Benzo(a)pyrene, Benzo(b)fluoranthene	June 1996 / Study ID #3
	ARSED1	Aroclor 1260, PCBS (total)	June 1995 / Study ID #1
	PSRSD1	Arsenic	September 1992 / Study ID#2
Water	1_14	Arsenic	May 1998 / Study ID #14
	2_14	p,p'-DDE, PCBS (total)	July 1998 / Study ID #14
	4A_14	Heptachlor (pesticide)	February 1998 / Study ID #14
	6_14	p,p'-DDT	February 1998 / Study ID #14

[1]Sample stations 4 and LA were nearly co-located

Study ID A1: MD Dept of Natural Resources Data. 1990. (Not available for review at the time this report was prepared.)

Study ID 1: Baker Environmental. ND. Washington Navy Yard. Baker Environmental. (Not available for review at the time this report was prepared.)

Study ID 2: Baker Environmental. ND. Bolling AFB - SW Corner Landfill. (Not available for review at the time this report was prepared.)

Study ID 3: ChemWorld Environmental. 1997. WA Gas, East Station Project, 1996. (Not available for review at the time this report was prepared.)

Study ID 4: Cummins, J.D. and D.J. Velinsky. 1993. 1992 D.C. fish tissue analysis for the evaluation of human health risks. District of Columbia, Department of Consumer and Regulatory Affairs, Water Quality Control Branch, Water Resources Management Division, Washington, D.C. 7 pages.

Study ID 5: Block, E. 1990. Organochlorine residues and histopathological examination of fish from the Potomac and Anacostia Rivers, Washington, DC. U.S. Fish and Wildlife Service, Environmental Contaminants Division, Annapolis, MD. AFO-C90-01 30 pages.

Table 6-7a. Category 3 - Detected Chemicals (sorted by chemical class and name)

CAS No.	Chemical Name	Chemical Class
95943	1,2,4,5Tetrachlorobenzene	ABN
120821	1,2,4Trichlorobenzene	ABN
95501	1,2Dichlorobenzene	ABN
541731	1,3Dichlorobenzene	ABN
106467	1,4Dichlorobenzene	ABN
95954	2,4,5Trichlorophenol	ABN
88062	2,4,6Trichlorophenol	ABN
120832	2,4Dichlorophenol	ABN
105679	2,4Dimethylphenol	ABN
51285	2,4dinitrophenol	ABN
121142	2,4Dinitrotoluene	ABN
606202	2,6Dinitrotoluene	ABN
78933	2Butanone	ABN
91587	2Chloronaphthalene	ABN
95578	2Chlorophenol	ABN
591786	2Hexanone	ABN
95487	2Methylphenol	ABN
88744	2Nitroaniline	ABN
88755	2Nitrophenol	ABN
91941	3,3'Dichlorobenzidine	ABN
99092	3Nitroaniline	ABN
101553	4Bromophenyl phenyl ether	ABN
59507	4Chloro3methylphenol	ABN
106478	4Chloroaniline	ABN
7005723	4Chlorophenyl phenyl ether	ABN
108101	4Methyl2pentanone	ABN
106445	4Methylphenol	ABN
100016	4Nitroaniline	ABN
100027	4Nitrophenol	ABN

Table 6-7a. Category 3 - Detected Chemicals (sorted by chemical class and name)

CAS No.	Chemical Name	Chemical Class
67641	Acetone	ABN
71432	Benzene	ABN
65850	Benzoic acid	ABN
100516	Benzyl alcohol	ABN
111911	Bis(2chloroethoxy)methane	ABN
111444	Bis(2chloroethyl)ether	ABN
39638329	Bis(2chloroisopropyl) ether	ABN
85687	Butylbenzyl phthalate	ABN
108907	Chlorobenzene	ABN
132649	Dibenzofuran	ABN
84662	Diethyl phthalate	ABN
131113	Dimethyl phthalate	ABN
84742	Dinbutyl phthalate	ABN
100414	Ethylbenzene	ABN
87683	Hexachlorobutadiene	ABN
77474	Hexachlorocyclopentadiene	ABN
67721	Hexachloroethane	ABN
75092	Methylene chloride	ABN
98953	Nitrobenzene	ABN
621647	NnitrosodiNpropylamine	ABN
86306	Nnitrosodiphenylamine	ABN
608935	Pentachlorobenzene	ABN
87865	Pentachlorophenol	ABN
108952	Phenol	ABN
100425	Styrene	ABN
108883	Toluene	ABN
95476	Xylene, ortho	ABN
1330207	Xylenes, total	ABN
11097691	Aroclor 1254	PCB
55722275	TCDF, total	CDF
7429905	Aluminum	METAL
7440360	Antimony	METAL
7440393	Barium	METAL
7440417	Beryllium	METAL
7440428	Boron	METAL
16065831	Chromium III	METAL
18540299	Chromium VI	METAL
7.44047e+006	Chromium, total	METAL
7440484	Cobalt	METAL
7440508	Copper	METAL
57125	Cyanide	METAL
7439896	Iron	METAL
7439965	Manganese	METAL
7440020	Nickel	METAL
7782492	Selenium	METAL

Table 6-7a. Category 3 - Detected Chemicals (sorted by chemical class and name)

CAS No.	Chemical Name	Chemical Class
7440224	Silver	METAL
7440246	Strontium	METAL
7440315	Tin	METAL
7440622	Vanadium	METAL
7440666	Zinc	METAL
22967926	Methylmercury	ORGANOMETAL
91576	2Methylnaphthalene	PAH
83329	Acenaphthene	PAH
120127	Anthracene	PAH
207089	Benzo(k)fluoranthene	PAH
92524	Biphenyl	PAH
218019	Chrysene	PAH
206440	Fluoranthene	PAH
86737	Fluorene	PAH
193395	Indeno(1,2,3c,d)pyrene	PAH
91203	Naphthalene	PAH
129000	Pyrene	PAH
115322	Dicofol	PESTICIDE
1031078	Endosulfan sulfate	PESTICIDE
72208	Endrin	PESTICIDE
319857	Hexachlorocyclohexanebeta	PESTICIDE
78591	Isophorone	PESTICIDE
2385855	Mirex (dechlorane)	PESTICIDE
2921882	Chlorpyrifos	PESTICIDE
1861321	Dacthal	PESTICDE
534521	4,6dinitro2methylphenol	UNCLASSIFIED

Category 3 chemicals that have been detected in the tidal Anacostia River and their maximum concentrations do not exceed an RBC; however, limitations in sample numbers or geographic distribution of sampling do not support a Not COPC classification. ABN, acid/base/neutral extractable; CDF, chlorinated dibenzofuran; PAH, polycyclic aromatic hydrocarbon; PCB, polychlorinated biphenyl; PEST, pesticide

Table 6-7b. Category 3 - *Not Detected* Chemicals (sorted by chemical class and name)

CAS No.	Chemical Name	Chemical Class
71556	1,1,1Trichloroethane	ABN
79345	1,1,2,2Tetrachloroethane	ABN
79005	1,1,2Trichloroethane	ABN
75343	1,1Dichloroethane	ABN
75354	1,1Dichloroethene	ABN
107062	1,2Dichloroethane	ABN
540590	1,2Dichloroethene	ABN
78875	1,2Dichloropropane	ABN
108601	2,2'Oxybis(1chloropropane)	ABN
118796	2,4,6Tribromophenol	ABN

Table 6-7b. Category 3 - *Not Detected* Chemicals (sorted by chemical class and name)

CAS No.	Chemical Name	Chemical Class
110758	2Chloroethylvinyl ether	ABN
92875	Benzidine	ABN
75274	Bromodichloromethane	ABN
75252	Bromoform	ABN
74839	Bromomethane	ABN
86748	Carbazole	ABN
75150	Carbon disulfide	ABN
56235	Carbon tetrachloride	ABN
75003	Chloroethane	ABN
67663	Chloroform	ABN
74873	Chloromethane	ABN
156592	cis1,2Dichlorethene	ABN
10061015	cis1,3Dichloropropene	ABN
124481	Dibromochloromethane	ABN
62759	Nnitrosodimethylamine	ABN
127184	Tetrachloroethylene	ABN
156605	Trans1,2Dichloroethene	ABN
10061026	trans1,3Dichloropropene	ABN
79016	Trichloroethene	ABN
108054	Vinyl acetate	ABN
75014	Vinyl chloride	ABN
11104282	Aroclor 1221	PCB
11141165	Aroclor 1232	PCB
12672296	Aroclor 1248	PCB
12674112	Aroclor 1016	PCB
53469219	Aroclor 1242	PCB
7439987	Molybdenum	METAL
7440280	Thallium	METAL
	Monobutyl tin	ORGANOMETAL
959988	Endosulfanalpha	PESTICIDE
7421934	Endrin aldehyde	PESTICIDE
53494705	Endrin ketone	PESTICIDE
72435	Methoxychlor	PESTICIDE
8001352	Toxaphene	PESTICIDE

Category 3 chemicals that have not been detected in tidal Anacostia, however, limitations in sample numbers or geographic distribution of sampling do not support a COPC classification. ABN, acid/base/neutral extractable; PCB, polychlorinated biphenyl

Table 6-8. Category 4 Chemicals (sorted by chemical class and name)

CAS No.	Chemical Name	Chemical Class
132650	Dibenzothiophene	ABN
1002535	Dibutyl tin	ABN

26601649	Hexachlorobiphenyl	ABN
20763886	Tributyl tin	ABN
22569728	Arsenic III	METAL
	Dimethylarsenic	ORGANOMETAL
	Monomethylarsenic	ORGANOMETAL
90120	1Methylnaphthalene	PAH
832699	1Methylphenanthrene	PAH
2245387	2,3,5Trimethylnaphthalene	PAH
581420	2,6Dimethylnaphthalene	PAH
208968	Acenaphthylene	PAH
192972	Benzo(e)pyrene	PAH
191242	Benzo(g,h,i)perylene	PAH
28804888	Dimethylnaphthalene	PAH
198550	Perylene	PAH
85018	Phenanthrene	PAH
28652779	Trimethylnaphthalene	PAH
5103731	cisNonachlor	PESTICDE
33213659	Endosulfanbeta[1]	PESTICDE
319868	Hexachlorocyclohexanedelta[1]	PESTICIDE
53190	o,p'DDD[1]	PESTICDE
3424826	o,p'DDE[1]	PESTICIDE
789026	o,p'DDT[1]	PESTICIDE
27304138	Oxychlordane	PESTICIDE
39765805	Trans nonachlor	PESTICIDE
5103742	Transchlordane[2]	PESTICIDE
5103719	Cischlordane[2]	PESTICIDE
5566347	Gammachlordane[2]	PESTICIDE
	BHCs, total	PESTICIDE
1825214	Pentachloroanisole	UNCLASSIFIED

Category 4 includes chemicals that have been detected in the tidal Anacostia and for which RBCs were not available. ABN, acid/base/neutral extractable; PAH, polycyclic aromatic hydrocarbon

[1]Chemicals for which an ARAR value for fish tissue was available; the maximum concentration was less than the ARAR value.

[2]Chemicals for which an ARAR value for fish tissue was available; the maximum concentration exceeded the ARAR value and the chemical was also placed in Category 1.

REFERENCES FOR CHAPTER 6

Government of the District of Columbia, Department of Consumer and Regulatory Affairs. 1994. Water Quality Standards. March 4. 41D.C. Reg.1075.

Maryland Department of the Environment. 2000. Water Quality Criteria for Toxic Substances. Available from: http://www.mde.state.md.us/wqstandards/toxics1.html-toxics8.html. June 3.

NOAA. 2000. Anacostia River Watershed Database and Mapping Project. Release 1. National Oceanic and Atmospheric Administration, Office of Response and Restoration, Coastal Protection and Restoration Division.

U.S. EPA. 1991. Maximum Contaminant Level Goals and National Primary Drinking Water Regulations for Lead and Copper. FR Vol 56 No. 110 June 7 p. 26460-91.

U.S. EPA. 1996. Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil. Publication 9285.7-081

U.S. EPA. 1997. The Incidence and Severity of Sediment Contamination in Surface Waters of the United States. Vol 1: National Sediment Quality Survey. Office of Science and Technology. EPA 823-R-97-006.

U.S. EPA. 1998. National Recommended Water Quality Criteria. December 10. 63(237)FR68354-68364.

U.S. EPA. 1999. Risk-Based Concentration Tables. Memorandum from Jennifer Hubbard, Region III U.S. EPA to RBC Table Users.

7. ECOLOGICAL RISK SCREENING

7.1 INTRODUCTION

The goal of this screening-level ecological risk assessment (SLERA) is to evaluate risk associated with contaminants present in the water column and sediments throughout the lower Anacostia River. The identification and assessment of specific sources of contamination within this area were not considered as part of this assessment.

This assessment was conducted in accordance with U.S. Environmental Protection Agency Risk Assessment Guidance (U.S. EPA, 1997). This report of findings is organized into ten sections. Section 7.2 presents results from the problem formulation, including discussions of resources and risk; selection of contaminants of potential concern (COPCs); the fate, transport, and ecotoxicity of COPCs; selection of receptors of concern (ROCs); identification of exposure pathways; and assessment and measurement endpoints. Section 7.3 is an evaluation of risk to benthic invertebrates, Section 7.4 is an evaluation of risk to fish, Section 7.5 is an evaluation of risk to birds, and Section 7.6 is an evaluation of risk to mammals. Section 7.7 is a qualitative analysis of the uncertainty in these risk evaluations. Section 7.8 provides the results of additional evaluations that were conducted. A summary of the results of risk evaluations conducted as part of the SLERA are provided in Section 8.3. Recommendations with regard to further work that could be conducted as part of a baseline risk assessment or an evaluation of remedial options are provided in Section 9.

Ecological risk assessment is a process whereby the likelihood that adverse biological impacts are occurring or may occur as a result of exposure to one or more stressors (U.S. EPA, 1992) is evaluated. Screening level risk assessments are simplified evaluations to decide whether further investigations are warranted or not. These screenings can be done quickly because they can be based upon existing data with generic or standard, conservative assumptions applied for many of the parameters involved in estimating the mobility and toxicity of contaminants of concern (U.S. EPA, 1997). When the stressors of concern are toxic substances, these assumptions are appropriate because of the conservative bias that must be applied to these types of evaluations. For investigations of sites where toxic hazardous materials have been released, it is important to minimize the probability of a conclusion that the site poses no risk when in fact it does (the so-called false negative outcome). To ensure that sites which could pose an ecological risk are thoroughly evaluated, the focus of the initial screening is to determine whether data exist to conclusively prove that a site does not pose significant risk. Although the standard, conservative assumptions applied are often considered unrealistic, a strong bias in the direction of overestimating risk minimizes the probability of a false negative conclusion. If a site is shown to present a risk, or sufficient data does not exist to eliminate the potential for risk, then the site continues through a more rigorous evaluation process which refines the estimations of actual risk.

For the screening ecological risk assessment, assessment endpoints are chosen that represent the significant ecological functions valued by society. These assessment endpoints include adverse ecological effects of organisms that are exposed to the contaminants of concern, i.e., receptors, as determined according to the conceptual model developed for how contaminants move throughout the system. The assessment endpoints are typically expressed in terms of ensuring the viability of a population of species of interest or species that is representative of groups of organisms of interest. If threatened or endangered species are an assessment endpoint, then impacts to individuals (versus populations) becomes more critical. The estimation of risk for these assessment endpoints is made upon

the measurement endpoints. Measurement endpoints are the measurable ecological characteristics that are directly related to the condition of an assessment endpoint. For instance, measurements of survival and of normal reproduction and juvenile development are measurable parameters which contribute to an understanding of the long-term viability of a population. An ecological risk may have multiple assessment endpoints and there maybe multiple measurement endpoints for each individual assessment endpoint.

7.2 ENVIRONMENTAL SETTING AND CONTAMINANTS

7.2.1 SITE HISTORY

The natural and human use history of the Anacostia River watershed is described in Section 2. Aspects of these topics that are particularly relevant to the ecological assessment are summarized here. Land use in the Anacostia watershed is typical for major urban areas with high density development concentrated near the urban center. The average impervious surface coverage of the lower tidal river watershed is 27%. This leads to large influx of street runoff, non-point source inputs, and combined storm sewer inputs to the river. There is a large area of open space on National Park Service land adjacent to the tidal river. However, this land is managed as urban parks and, therefore, does not provide the natural vegetation and canopy cover usually associated with stream buffers.

The loss of tidal wetlands along the river has been a major factor in the degradation of aquatic habitat. The structure of the tidal river system has been dramatically altered over time to manage the massive sediment inputs historically generated by upstream development and agricultural erosion. This alteration has occurred through seawall construction, navigational dredging of the mainstem, and associated filling. These actions have collectively led to the destruction of the river's once-thriving fringe wetlands. The Army Corps of Engineers estimates that approximately 2,500 acres of tidal emergent wetlands have been destroyed in the lower Anacostia. This represents an overall loss of more than 90% of the originally-occurring tidal wetlands from the river. Even with the creation of 32 acres at Kenilworth Marsh, less than 100 acres of tidal emergent wetlands still exist.

7.2.2 SITE DESCRIPTION

This assessment is focused on the mainstem of the lower, tidal Anacostia River and its associated aquatic habitats. For the purposes of this screening risk assessment, the tidal Anacostia River is defined as the river proper, including the associated tideplains, floodplains, and wetlands, extending from the confluence of the Northeast and Northwest Branches to the confluence with the Potomac River, including the Kenilworth Marsh and Kingman Lake embayments. The entire study area is freshwater, but is tidally influenced with an average amplitude of 2.8 feet (Velinsky et al., 1992). The biological communities and habitats present within the study area suggest that the river can be divided into three separate zones—the lower river zone, upper river zone, and the Washington Ship Channel/Tidal Basin zone (Figure 7-1).

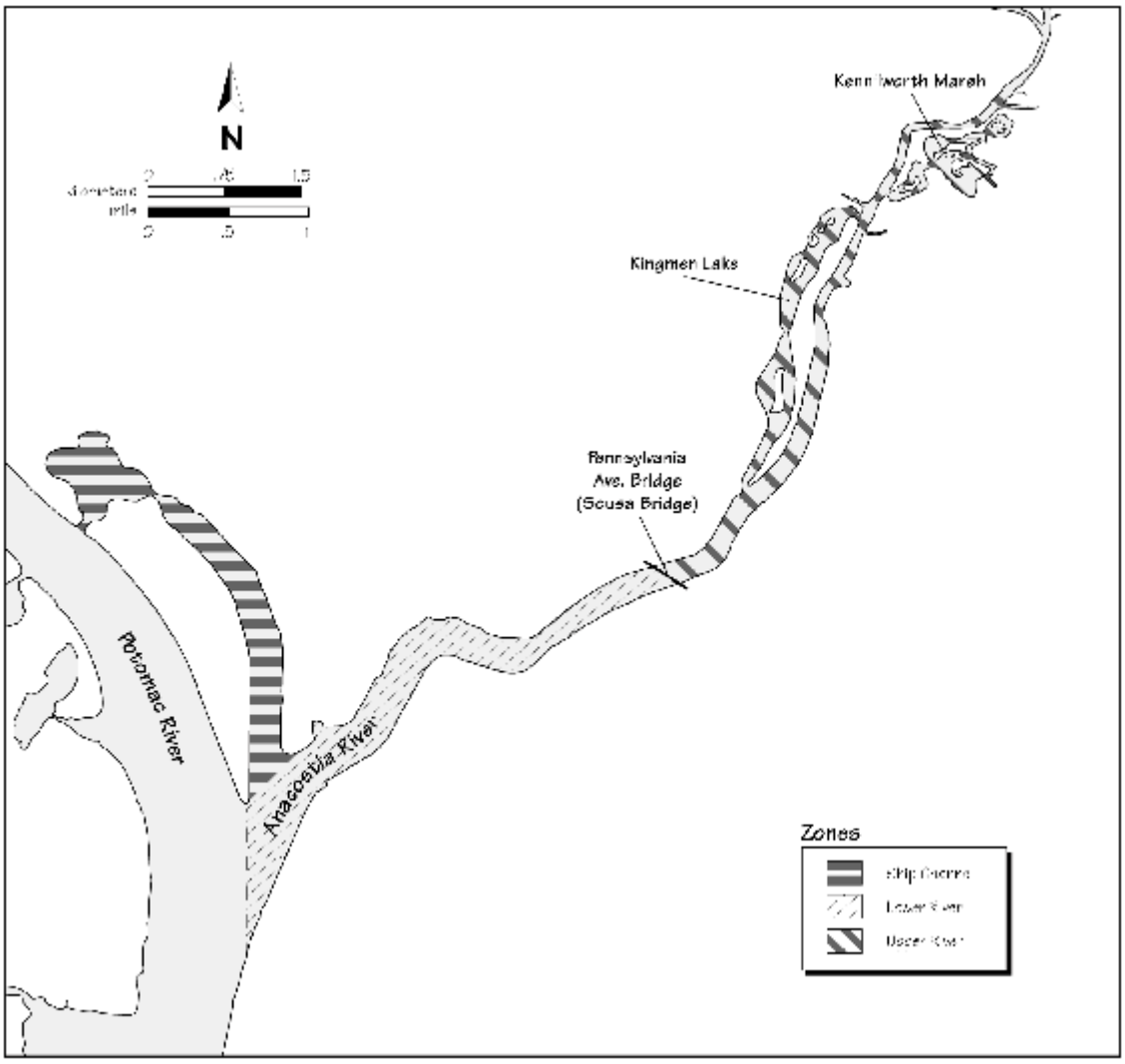

N
Kenilworth Marsh
Kingman Lake
Pennsylvania
Ave. Bridge
(Sousa Bridge)
Potomac River
Anacostia River
Zones

Figure 7-1. Delineation of zones for estimation of exposure for the ecological risk characterization.

The lower river zone extends about 4 miles from the river mouth to the Sousa Bridge (at Pennsylvania Avenue). The mouth of the Anacostia joins the Potomac River at Hains Point in Washington, D.C., 108 miles upstream from the Potomac River's mouth to the Chesapeake Bay. Sixty-two percent of the lower tidal Anacostia drainage area of 4,561 acres is in the District of Columbia, with the remaining 38% in Prince George's County.

The river in the lower zone is wide and channelized. This section of the river contains the federal navigation channel. The navigation channel is authorized for 300 to 600 feet wide, through much of this zone, and a depth of 16 feet at the mouth, dropping to 13 feet, and then reduced again to 6 feet above the Anacostia Bridge (at 11th Street).

The upper river zone extends from the lower river boundary for about 4.5 miles to the confluence of the Northwest and Northeast Branches, in the vicinity of Bladensburg, Maryland. This section of the river is less channelized, variable in width, and shallow, ranging from <1 to 15 feet in depth (Velinsky et al., 1992). The upper river zone also includes the Kenilworth Marsh, a 75 acre wetland which is the last remaining tidal freshwater wetland in the Washington D.C. area. Kingman Lake is also a part of the upper river zone. The lake is a channel to the river, running parallel for about 2 miles. The island separating the lake from the river was formed with dredge material. Like the upper river, the lake is shallow with average depths ranging from 5 to 10 feet. The upper river zone also contains the Anacostia federal navigation channel, which is currently being dredged.

The Washington Ship Channel is a northward trending channel, running parallel to the Potomac River for about a mile. The mouth of the channel is along the northern shoreline of the Anacostia River, just at its mouth with the Potomac. The Washington Ship Channel is about 500 feet wide and is periodically dredged to a depth of about 10 feet. The Tidal Basin connects with the upper end of the Ship Channel and with the Potomac River through culverts. The basin has a surface area of about 100 acres, and an average depth of 6 feet (Velinsky et al., 1992).

7.2.3 HABITAT DESCRIPTION

The Anacostia River watershed encompasses the south-eastern half of the District of Columbia and stretches northward into Prince George and Montgomery counties of Maryland. The entire watershed drains approximately 122 square miles. There are three major drainage areas comprising the Anacostia watershed: the Northwest Branch, the Northeast Branch, and the tidal drainage.

The entire watershed is an ecologically diverse system which contains both free-flowing and freshwater tidal segments. The watershed also covers two distinct geologic features: the Piedmont province which is characterized by relatively narrow and steep-sloped valleys of moderately thin soils, and the Coastal Plain which has gentler, more-rolling hillsides comprised of deeper sedimentary soil complexes. The Coastal Plain tends to support broader meandering streams than the Piedmont feature. The tidal drainage area does have small, non-tidal streams that flow directly to the tidal river, though most of these streams are enclosed in storm sewer systems.

Very little natural aquatic habitat is present in the lower river and Ship Channel zones. Most shorelines are composed of seawalls, docks, piers, and marinas (Herson-Jones et al., 1994; Velinsky et al., 1992). The upper river zone contains less channelized areas then the lower, but natural habitats are still substantially reduced. Lake Kingman was created by dredge spoils in the 1920s and is surrounded by park land and a golf course. The Kenilworth Marsh occupies approximately 75 acres on the east shore of the river immediately upstream of Kingman Lake. Much of the marsh has been restored as an emergent tidal freshwater wetland. Small areas of emergent wetlands are present near the river shore upstream of Kenilworth Marsh to the Northwest and Northeast Branches (Velinsky et al., 1992; NPS, 2000)
The Anacostia River can be classified as a warm-water stream with mean temperatures ranging from 3° C

(Celsius) in January to 26° C in August. Summer temperatures range from 18 to 32° C and can be an environmental stressor to some fish species. Similarly, dissolved oxygen concentrations during the summer months have been reported at below water quality standards of 5 mg/L. Water in the study area has a long residence time (~35 days) due to the large volume-to-flow ratio. The long residence time and the tidal nature of the river allow the substantial deposition of suspended sediments in the study area (Herson-Jones et al., 1994).

The following sections provide descriptions of the primary biological communities present in the aquatic habitats of the study area. A benthic, macroinvertebrate community associated with fine-grained, organically enriched sediments is present in the lower Anacostia River, as well as populations of benthic and pelagic fish and aquatic birds and mammals.

7.2.3.1 MACROINVERTEBRATE COMMUNITY

Sediment and the surface of submerged objects, such as rocks and pilings, provide habitat for many species of bottom-dwelling animals known as benthic organisms. A healthy community of benthic animals is usually dominated by a large number of small invertebrates (worms, amphipods, etc.) from diverse groups of animal families. The organisms that live below the sediment surface, called infauna, have a significant role in the riverine ecosystem as prey for larger animals, such as fish and crabs, and in cycling of nutrients. Larger, more visible and often more mobile invertebrates, such as crab, shrimp, and clams, are another substantial component of a benthic community. A macroinvertebrate community associated with fine-grained sediments highly enriched with organic matter is present in the lower Anacostia River. These animals are critical, not only because of their role in the ecosystem, but also because of the way that they feed often exposes them to sediment-associated contaminants. Many infaunal species ingest organic material deposited on the bottom, as part of the sediment. Most feed selectively on a particular size range of particles which have relatively higher levels of organic matter. These same organic-rich particles are those which are typically, also relatively, concentrated with contaminants.

Benthic studies, conducted in association with sediment investigations, collected benthic samples within the Anacostia River, Washington Ship Channel, and Tidal Basin. The number of benthic species (or species groups) observed ranged from 5 to 12 per station. Pollution-tolerant oligochaetes (freshwater worms) constituted 73 to 96 percent of the taxa observed at 8 of 9 stations within the study area. Chironomids (midges), another pollution-tolerant group was the second most abundant taxa. Together, the two groups of animals composed between 96 and 99 percent of all benthic organisms per station (Velinsky et al., 1992). Small numbers of mollusks (bivalves), amphipods (scuds), and other aquatic insects were also observed. The largest difference between the Ship Channel and the Tidal Basin was the percentage of chironomids in the sediments. Benthic samples collected in the Tidal Basin had a higher percentage of chironomids (mean = 44.5 percent) compared to the Ship Channel (mean = 12.5 percent) or the river (mean = 11.4 percent).

In general the benthic community within the study area can be characterized as having relatively few taxa, dominated by pollution tolerant species (Velinsky et al., 1992). The oligochaetes are considered the most pollution tolerant group of freshwater macroinvertebrates. The chironomids are also a pollution tolerant group compared to many taxa of aquatic insects and crustaceans. Oligochaetes and chironomids generally occupy habitats of very fine sediments with high organic content. The two groups are relatively less sensitive to organic and inorganic contamination and oxygen depletion (U.S. EPA, 1990; Burton, 1991).

7.2.3.2 FISH COMMUNITY

The finfish of the Anacostia River are among the more notable, more visible species of the river because of their commercial and recreational or subsistence value. Many of those fish that are not directly fished by humans may serve as prey for predatory species. Since many fish are near the top of a food web, and therefore rely on the health and abundance of all the food web levels below them, the general health of the fish community is often a broad indicator for the general state of a riverine ecosystem.

At any given time, the fish community in a river may be comprised of year-round residents of any age class, adults which are spawning in the area or adults which are migrating through the area to reach their spawning grounds, early life stages which use the area as a nursery ground, and various age classes of fish which are feeding within the area. There are seasonal patterns for many fish, linked to either their reproductive cycle or their habitat preference, e.g., a move to warmer waters during winter. The distribution of fish is also partially determined by their living requirements, primarily as determined by salinity and temperature.

Finfish surveys have been conducted throughout tidal portions of the Anacostia River. A recent fish survey cited by Herson-Jones et al. (1994) reported 25 species in the river, of which 11 species comprised over 98 percent of the total (Table 7-1). Survey data indicate that the study area provides substantial spawning and nursery habitat for the anadromous *Alosids* (river herring and shads). Blueback herring and alewife dominate the fish community, particularly in the lower river. Other studies have shown that juvenile blueback herring concentrate near the confluence of the Anacostia and Potomac Rivers, suggesting that this area may be a nursery for the species (Herson-Jones et al., 1994). Alewife and blueback herring migrate and spawn in the study area from late-March to mid-May, with most spawning adults gone by mid-summer. Eggs hatch in 2 to 6 days depending upon temperature (Scott and Crossman, 1973) and larvae and juveniles use the study area as a nursery through September before out-migrating to Chesapeake Bay and the Atlantic Ocean (Lippson et al., 1980). One other *Alosid* present in substantial numbers was the gizzard shad, comprising between 2 and 10 percent of the total number of fish collected in the lower and upper river (Herson-Jones et al., 1994).

The anadromous white perch was the most frequently encountered species, occurring in nearly 70 percent of all samples collected in the lower and upper Anacostia River. White perch were distributed fairly evenly throughout the study area, relative to the total number of fish collected (Herson-Jones et al., 1994). Lippson et al. (1980) reported that the study area is a secondary spawning ground for the species. White perch are also spring spawners, spawning in the study area in April and May. Juveniles use the area as a nursery through October before out-migrating to estuarine portions of the Potomac River and Chesapeake Bay (Lippson et al., 1980).

Three freshwater residents, pumpkinseed, brown bullhead, and spottail shiner, were common in samples collected in the upper river. In the upper river, three euryhaline species, inland silversides, mummichog, and banded killifish, were observed in lower numbers. In the lower river, all of these species were observed at one percent or less in total abundance because of the dominance of the *Alosids* (Table 7-1). One other anadromous species, the striped bass, was found at less than one percent abundance in both the upper and lower river (Herson-Jones et al., 1994). This species is not known to spawn in the Anacostia River, so it would not be expected in large numbers (Lippson et al., 1980).

Table 7-1. Finfish Species and Composition Observed in a Recent Fish Survey in the Anacostia River (Herson-Jones et al. 1994)

Fish Species	Scientific Name	Percent Total Lower River	Percent Total Upper River	Percent Total Entire Study Area
Anadromous Species				
Blueback herring/ Alewife*	Alosa spp.	75	26	63
White perch	Morone americana	18	19	18
Gizzard shad	Dorosoma cepedianum	2	10	4
Striped bass	Morone saxatilis	0.7	0.1	0.5
Estuarine/Euryhaline Species				
Banded killifish	Fundulus diaphanus	0.3	7	4
Inland silverside	Menidia berylina	1	2	1
Mummichog	Fundulus heteroclitus	0.2	5	1
Freshwater Resident Species				
Pumpkinseed	Lepomis gibbosus	0.9	12	4
Brown bullhead	Ameiurus nebulosus	0.6	14	2
Spottailed shiner	Notropis hudsonius	0.6	4	1
Other species		0.7	0.9	1.5

* Blueback herring and alewife were not separated in the study

Several other freshwater species not observed in the fish survey have been reported in the Anacostia watershed. These include the non-parasitic least brook and American brook lampreys (*Lampetra aepyptera* and *L. appendix*), chain pickerel (Esox niger), mosquito fish (*Gambusia affinis*), large and smallmouth bass (*Micropterus salmoides* and *M. dolomieu*), black crappie (*Pomoxis nigromaculatus*), and several other sunfish species (*Lepomis* spp.). These species are generally more abundant in the upper watershed upstream of the study area and in tributary streams, where the degree of habitat modification and degradation is not as extensive (Lippson et al., 1980).

Other anadromous species reported in the watershed include the sea lamprey (*Petromyzon marinus*), American shad (*Alosa sapidissima*), Atlantic sturgeon (*Acipenser oxyrhynchus*), and the Federally endangered shortnose sturgeon (*A. brevirostrum*). Estuarine species include the hogchoker (*Trinectes maculatus*), which can be found as far upstream as the Washington D.C. area (Lippson et al., 1980).

7.2.3.3 AQUATIC BIRDS

The National Parks Service has listed 188 species of terrestrial, riparian, and aquatic birds in the lower Anacostia watershed, of which over 50 are associated with the aquatic environment (Table 7-2; NPS 2000). The habitats in the study area are used by aquatic birds that are year round residents, by local breeding populations, and by highly migratory species that either overwinter in the area or pass through on the way to northern or southern destinations. Most breeding areas are limited to the Kenilworth Marsh, Kenilworth Park, and Kingman Lake. Outside of these areas, much of the mainstem of the Anacostia River, Washington Ship Channel, and Tidal Basin have developed shorelines and are only used for foraging. The habitat use and feeding strategies of the aquatic birds in the lower Anacostia are summarized in Table7-2.

Table 7-2. Aquatic Birds Documented Within the Lower Anacostia River Watershed, Habitat Use and Feeding Strategy

Common Name	Scientific Name	Habitat Use			Feeding Strategy
		Resident	Overwinter	Breeding	
Duck-Like Birds					
Bufflehead	Bucephala albeola				Omnivore
Canvasback	*Aythya valisineria*				Grazer
Gadwall	*Anas strepera*				Omnivore
Goldeneye	*Bucephala clangula*				Invertebrates
Mallard	*Anas platyrhynchos*				Omnivore
Oldsquaw	*Clangula hyemalis*				Invertebrates
Pintail	*Anas acuta*				Omnivore
Ringneck duck	*Aythya collaris*				Grazer
Northern shoveler	*Anas clypeata*				Omnivore
Ruddy duck	*Oxyjura jamaicensis*				Grazer
Blue-winged teal	*Anas discors*				Omnivore
Green-winged teal	*Anas crecca*				Omnivore
American widgeon	*Anas americana*				Grazer
Wood duck	*Aix sponsa*				Grazer
Canada goose	*Branta canadensis*				Grazer
Snow goose	*Chen caerulescens*				Grazer
Common merganser	*Mergus merganser*				Piscivore
Hooded merganser	*Lophodytes cucullatus*				Invertebrates
Red-breasted merganser	*Mergus serrator*				Piscivore
American coot	*Fulica americana*				Grazer
Eared grebe	*Podiceps nigricollis*				Piscivore

Table 7-2. Aquatic Birds Documented Within the Lower Anacostia River Watershed, Habitat Use and Feeding Strategy

Common Name	Scientific Name	Habitat Use			Feeding Strategy
		Resident	Overwinter	Breeding	
Horned grebe	*Podiceps auritus*				Piscivore
Pied-billed grebe	*Podilymbus podiceps*				Piscivore
Red-necked grebe	*Podiceps grisegena*				Piscivore
Common loon	*Gavia immer*				Piscivore
Red-throated loon	*Gavia stellata*				Piscivore
Sora rail	*Porzana carolina*				Omnivore
Virginia rail	*Rallus limicola*				Omnivore
Common gallinule	*Gallinula chloropus*				Omnivore
Wading Birds					
American bittern	*Botaurus lentiginosus*				Piscivore/ Invertebrates
Least bittern	*Ixobrychus exilis*				Piscivore/ Invertebrates
Cattle egret	*Bubulcus ibis*				Invertebrates
Great egret	*Casmerodius albus*				Invertebrates
Snowy egret	*Egretta thula*				Invertebrates
Black-crowned night heron	*Nycticorax nyticorax*				Piscivore/ Invertebrates
Great blue heron	*Ardea herodias*				Piscivore
Green heron	*Butorides virescens*				Piscivore/ Invertebrates
Little blue heron	*Egretta caerulea*				Piscivore/ Invertebrates
Gulls and Terns					
Herring gull	*Larus argentatus*				Omnivore
Laughing gull	*Larus atricilla*				Piscivore
Ring-billed gull	*Larus delawarensis*				Omnivore
Caspian tern	*Sterna caspia*				Piscivore
Forsters tern	*Sterna forsteri*				Piscivore
Least tern	*Sterna antillarum*				Piscivore
Sandpipers					
Dunlin	*Calidris alpina*				Invertebrates

Table 7-2. Aquatic Birds Documented Within the Lower Anacostia River Watershed, Habitat Use and Feeding Strategy

Common Name	Scientific Name	Habitat Use			Feeding Strategy
		Resident	Overwinter	Breeding	
Sanderling	*Calidris alba*				Invertebrates
Least sandpiper	*Calidris minutilla*				Invertebrates
Pectoral sandpiper	*Calidris melanotos*				Invertebrates
Semipalmated sandpiper	*Calidris pusilla*				Invertebrates
Solitary sandpiper	*Tringa solitaria*				Invertebrates
Spotted sandpiper	*Acitis macularia*				Invertebrates
Stilt sandpiper	*Calidris himantopus*				Invertebrates
Blackbirds					
Red-ringed blackbird	*Agelaius phoeniceus*				Omnivore
Rusty blackbird	*Euphagus carolinus*				Omnivore
Other Species					
Double-crested cormorant	*Phalacrocorax auritus*				Piscivore
Belted kingfisher	*Ceryle alcyon*				Piscivore
Osprey	*Pandion haliaetus*				Piscivore

The largest group of aquatic birds present in the study area are the duck-like species within the families *Anatidae* (ducks and geese), *Gaviidae* (loons), *Podicipedidae* (grebes), and *Rallidae* (coots and rails). Nearly 30 species represent these four families in the study area, most of which are associated with the Kenilworth Marsh, Kingman Lake, and the mainstem Anacostia River in the upper river zone (NPS 2000).

The ducks, geese, coots, and rails are largely grazers and omnivorous. Canvasback, ringnecked duck, ruddy duck, widgeon, wood duck, Canada goose, and snow goose are primarily grazers of aquatic and terrestrial vegetation. Several other species, such as mallards, goldeneye, bufflehead, oldsquaw, and common gallinule, are omnivorous feeding on vegetation, insects, and small aquatic invertebrates. The mergansers, loons, and grebes are strong divers and swimmers and feed on fish and aquatic invertebrates (Udvardy and Farrand, 1998).

The ducks and geese primarily use the study area for overwintering, although a few species such as wood duck, mallard, and rails may breed during the spring and summer in the upper zone (NPS 2000; Udvardy and Farrand, 1998).

Nine species of wading birds within the family *Ardeidae*, which includes the herons, bitterns, and egrets,

have been documented in less developed shoreline habitats in the study area (NPS 2000). The large great blue heron is primarily a piscivore while the smaller herons, bitterns, and egrets feed on fish, frogs, crustaceans, other aquatic invertebrates, and insects. Most of the wading birds are permanent residents of the study area, although cattle egrets are largely an inland species that breed near water (Udvardy and Farrand, 1998).

Three species of gulls and three species of terns, within the family *Laridae*, have been documented in the study area (NPS 2000). The laughing gull is a piscivore while the herring gull is a scavenging omnivore. Both are permanent residents of the region. The ring-billed gull only overwinters in the study area, breeding inland. The terns are primarily piscivores with the exception of the small least tern, which also feeds on aquatic invertebrates. The terns and gulls are colony breeders with most breeding in the region, but it is not known if colonies are present within the study area (Udvardy and Farrand, 1998).

Eight species of sandpiper within the family *Scolopacidae* have been documented in the study area (NPS 2000). Most of the sandpipers breed in the Arctic or sub-Arctic and overwinter in central to South America, so are transient within the study area. The exception is the spotted sandpiper which breeds in the region. The sanderling and dunlin also overwinter in the region, but usually occupy coastal beaches. All of the sandpipers feed primarily on benthic invertebrates found in shallow water sediments (Udvardy and Farrand, 1998).

Two species of blackbird within the family *Emberizidae* have been documented in the study area (NPS 2000). Both species are common year round residents of marshes and bogs of the upper zone. The blackbirds are omnivores, feeding on aquatic invertebrates, grains, and seeds (Udvardy and Farrand, 1998).

Three other important piscivores present within the study area include the osprey (family *Accipitridae*), kingfisher (family *Alcedinidae*), and double crested cormorant (family *Phalacrocoracidae*) (NPS 2000). The osprey is one of the few raptor species that has a strong association with water. The species is a permanent resident of the region, but any specimens that occupy the upper zone likely have a very large home range. Osprey feed almost exclusively on fish, although they have been observed on occasion taking other prey such as birds, frogs, and crustaceans (U.S. EPA, 1993; Udvardy and Farrand, 1998). The kingfisher is a permanent resident of the region, occupying areas of the Kenilworth Marsh and Kingman Lake in the upper river zone (NPS 2000). The kingfisher is highly piscivorous. The double crested cormorant is also a permanent resident of the region that breeds in both coastal and inland areas. The cormorant is also highly piscivorous (Udvardy and Farrand, 1998).

7.2.3.4 MAMMAL COMMUNITY

The National Parks Service has listed 17 species of mammals that reside in the entire Anacostia watershed, of which beaver, river otter, muskrat, mink, and raccoons are commonly-to-exclusively associated with aquatic environments (NPS 2000). As with the aquatic bird community, aquatic mammal populations are found primarily in the upper zone and Kenilworth Marsh.

Beaver (*Castor canadensis*) are almost exclusively aquatic, occupying rivers, streams, and wetlands. The species has been documented in the study area and is a likely common inhabitant of the upper zone (NPS 2000). Beaver are entirely herbivorous, most commonly consuming bark of certain hardwoods such as poplar, aspen, birch, cherry, willow, maple, and alder. Aquatic vegetation is also consumed. Beaver are active year round.

River otter (*Lutra canadensis*) are almost exclusively aquatic, occupying rivers, lakes, and other waters that show little human impact (U.S. EPA, 1993). The species has been documented within the study area, but is likely limited to less developed areas in the upper zone (NPS 2000). The species is primarily

piscivorous, but will opportunistically consume crustaceans, aquatic insects, amphibians, insects, birds, mammals, and turtles. River otter are active the whole year (U.S. EPA, 1993).

Muskrat (*Ondatra zibethicus*) inhabit freshwater streams, lakes, wetlands, ponds, brackish marshes, and salt marshes (U.S. EPA, 1993). They likely occupy surface waters of Kingman Lake, Kenilworth Marsh, and the upper zone of the Anacostia River (NPS 2000). Muskrats are primarily herbivorous, feeding on roots and basal portions of plants, as well as shoots, stems, and leaves. Omnivorous populations are also known to exist, supplementing vegetation with crayfish, fish, frogs, turtles, and young birds. Muskrats are active the whole year, using constructed dens to insulate themselves from summer heat and winter cold (U.S. EPA, 1993).

Mink (*Mustela vison*) are found associated with aquatic habitats of all kinds, including rivers, streams, lakes, and ditches, as well as wetlands, and backwater areas (U.S. EPA, 1993). Within the study area, they are most likely associated with the upper zone, Kenilworth Marsh, and Kingman Lake (NPS 2000). Mink are opportunistic predators, taking whatever prey is abundant. Mammals are the mink's most important prey in many parts of their range, but mink also hunt aquatic prey such as fish, amphibians, and crustaceans. The species is active year round (U.S. EPA, 1993).

Raccoons (*Procyon lotor*) are the most abundant and widespread medium-sized mammal in North America (U.S. EPA, 1993) and may be common within the study area (NPS 2000). The species is found near virtually every aquatic habitat, particularly various freshwater wetlands and salt marshes. The raccoon is an omnivorous and opportunistic feeder. They feed on fruits, nuts, grains, insects, frogs, crayfish, eggs, and virtually any animal and vegetable matter. The proportion of different foods in their diet depends on location and season, although plants are usually a more important component. They may focus on a preferred food when it is available. In the region of the study area, raccoons undergo a winter dormancy lasting up to four months (U.S. EPA, 1993).

7.2.4 ECOTOXICITY AND POTENTIAL RECEPTORS

7.2.4.1 CONTAMINANTS OF POTENTIAL CONCERN

COPCs were selected for each potential ecological receptor group. Reviewing the compiled sediment and tissue data for the Anacostia River identified five classes of contaminants: polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), dioxins and furans, chlorinated pesticides, and trace elements.

7.2.4.2 ECOTOXICITY OF THE CONTAMINANTS OF POTENTIAL CONCERN

7.2.4.2.1 POLYCYCLIC AROMATIC HYDROCARBONS

PAHs are a class of nonpolar organic compounds characterized by highly aromatic, fused-ring structures. Environmental sources of PAHs include petroleum, petroleum products, and combustion residues (i.e., soot particles). Because of their low water solubilities (0.0003 to 34 mg/L) and high octanol-water partition coefficients (log K_{ow} = 3.4 to 7.6), PAHs in aquatic systems tend to be associated with sediments and biota (Eisler, 1987).

In the Anacostia River, the fate and transport of PAHs will be largely controlled by sediment organic carbon content and dissolved organic carbon concentrations in the sediment porewater and water column. The extent to which an individual PAH compound will tend to be associated with either sediment or dissolved organic carbon depends on the relative hydrophobicity of the compound, which can be predicted from its molecular weight (Clement, 1985).

Low-molecular-weight PAHs (LPAHs), with three aromatic rings or less, are more water soluble and more easily degraded. High-molecular-weight PAHs (HPAHs), with 4 or more aromatic rings, will tend to persist in sediments where they are subjected to burial, resuspension, and degradation reactions. The available literature suggests that microbes degrade HPAHs slower than LPAHs. Half-lives for these compounds range from months to years. Furthermore, biodegradation probably occurs more slowly in aquatic systems than in soil (Clement Associates 1985).

PAHs vary substantially in their toxicity to aquatic organisms. LPAHs such as naphthalene, fluorene, phenanthrene, and anthracene are acutely toxic to aquatic organisms. Acute lethality increases with increasing alkyl substitution on the lower molecular weight compounds (Van Luik 1984). Many of the HPAHs, such as chrysene and benzo(a)pyrene, are less acutely lethal but demonstrably carcinogenic, mutagenic, or teratogenic to a wide variety of organisms including fish, amphibians, birds, and mammals (Moore and Ramamoorthy 1984; Eisler 1987). Among aquatic organisms, acute lethality is most pronounced among crustaceans and least among teleost fish (Eisler 1987).

Benthic Invertebrates – Effects of PAHs observed in benthic invertebrates include inhibited reproduction, delayed emergence, sediment avoidance, and mortality (Eisler, 1987; Landrum et al., 1991). In a study of PAH toxicity to the amphipod *Diporeia*, the mechanism identified as most likely responsible for observed acute toxic responses to PAHs was narcosis (Landrum et al., 1991). Generally, aquatic invertebrates are less able to metabolize PAHs than aquatic vertebrates, although metabolization rates vary widely within and between phyla (Meador et al., 1995). Thus, invertebrates tend to be susceptible to PAHs due to acute lethality by narcosis more so than for other organisms which actively metabolize these compounds.

Fish – Since PAHs are generally hydrophobic compounds, they must be metabolized to more water-soluble forms before they are excreted. In most fish, uptake of PAHs is rapid, but metabolism and excretion are too, so that concentrations found in tissues are generally low. The major route of elimination is through excretion into bile. The biotransformation and excretion rates can vary widely among fish species (Meador et al., 1995). Fish exposed to PAHs may be induced to produce higher levels of enzymes capable of transforming PAHs to more excretable, but occasionally more carcinogenic, metabolites (O'Connor and Huggett, 1988).

Because fish rapidly metabolize and excrete PAHs, fish tissue concentrations of the original, untransformed parent PAH compounds do not provide a useful measure of exposure to fish (Varanasi et al., 1989). Determining concentrations of PAHs in sediment is a useful measure of exposure because exposure to PAH-contaminated sediment has been linked to adverse effects in fish. These impacts include reproductive impairment, immune dysfunction, increased incidence of liver lesions, and other histopathological endpoints (Malins et al., 1987; Johnson et al., 1988; Varanasi et al., 1992, Baumann et al., 1996). Fin erosion and liver abnormalities have also been observed in fish exposed to extracts from PAH-contaminated sediments (Fabacher et al., 1991). Other studies report sublethal effects on the cellular immune system (reduced macrophage activities) in fish exposed to PAH-contaminated sediments, that could result in increased susceptibility to disease (Weeks and Warinner, 1984, 1986; Weeks et al., 1986). The most common diseases generally affect the liver, although cataracts and pollution-related disorders of the skin and gills may also occur (O'Connor and Huggett, 1988).

Birds – Very little data are available on the toxicity of PAHs in birds. In one study, Patton and Dieter (1980) fed mallards diets that contained 4,000 mg PAHs/kg for a period of 7 months. No mortality or visible signs of toxicity were evident during the exposure; however, liver weights increased 25 percent and blood flow to the liver increased 30 percent when compared to controls (Eisler, 1985). In addition, PAH mixtures applied to the surface of mallard eggs have been shown to result in increased embryo mortality and increased embryo deformation (Hoffman and Gay, 1981).

Mammals – In mammals, several PAH compounds have been shown to be potent carcinogens. In general, PAH carcinogens transform cells through genetic injury involving metabolism of the parent compound to a reactive diol epoxide (Eisler, 1985). In the case of benzo(a) pyrene, one isomer of the 7,8-diol, 9,10-epoxide is an exceptionally potent carcinogen to newborn mice and is believed to be the ultimate carcinogenic metabolite of this PAH (Slaga et al., 1978). One of the most toxicologically significant processes involved in response to PAH exposure is the interaction with drug metabolizing enzyme systems. Increased production of mixed-function oxidase enzymes in various small mammals has been induced by numerous PAH compounds (U.S. EPA, 1980). Interspecies differences in sensitivity to PAH-induced carcinogenesis are due largely to differences in levels of mixed function oxidase activities and these will affect rates at which active metabolites are converted to less active products (Neff, 1979).

7.2.4.2.2 POLYCHLORINATED BIPHENYLS

The fate and transport of PCBs in environmental systems is controlled by distribution or partitioning of PCBs between sediment, suspended particulates, surface water, and biota. The observed partitioning of nonionic organic chemicals, such as PCBs, is due to sorption to organic matter. The extent to which PCB congeners are associated with organic matter, relative to their dissolved aqueous concentrations, is related to their levels of chlorination. The more chlorinated congeners have stronger tendencies to be associated with particulate and dissolved organic matter than the less chlorinated congeners.

Bioaccumulation of PCBs occurs as a result of the partitioning of the congeners between an organism's tissues, particularly its lipids, and the ambient environment. Therefore, bioaccumulation is highly dependent on the organism's lipid content and trophic level, and on the hydrophobicity of the PCB congener. In addition, PCBs are subject to biomagnification through the food chain.

Although PCBs are generally persistent, they can be degraded *in situ* to a limited extent by resident microorganisms.

Benthic Invertebrates – PCBs have a wide variety of effects on aquatic organisms. There are significant interspecies differences in sensitivities to PCBs, even among species that are closely related taxonomically (Eisler 1986). Most studies of the effects of PCBs on benthic invertebrates have shown reproductive impairment and effects on survival and growth (Eisler, 1986).

Fish – Effects of PCBs on fish include mortality, growth-related impacts, behavior responses, biochemical alterations, and reproductive impairment. Of particular concern are the effects of dioxin-like PCB congeners, which have the same toxic mechanism as 2,3,7,8-TCDD (Walker and Peterson, 1991; Zabel et al., 1995). These dioxin-like PCB congeners cause early life stage mortality associated with blue-sac disease, which involves subcutaneous yolk sac edema (Wisk and Cooper, 1990; Walker et al., 1991).

In addition, numerous field studies have reported increased mortality, pathologic anomalies, and biochemical changes in feral fish collected from ecosystems where PCBs have been reported and correlated with the concentrations of PCBs in tissue (Niimi, 1996). These observations include reduced hatchability and poor survival of larvae taken from feral organisms and reared in the laboratory (Ankley et al., 1991; Mac and Schwartz, 1992). This impact is clearly important from an ecological perspective. Other impacts, such as behavioral responses and biochemical alterations, are more difficult to interpret, although some biochemical alterations may adversely affect reproduction (Sivarajah et al., 1978; Chen et al., 1986; Thomas, 1988).

Birds – A substantial amount of research has been conducted demonstrating adverse reproductive effects in piscivorous bird populations exposed to PCBs (Tillitt et al., 1992; Jones et al., 1993, 1994; Giesy et al.,

1994 a,b). The bulk of the research has focused on double-crested cormorants because deformities were first discovered in this species. Some work has been done to evaluate reproductive effects of PCBs in the great blue heron (Sanderson et al., 1994, 1997). Piscivorous birds display a number of symptoms similar to those observed in other avian species exposed to planar halogenated hydrocarbons in the laboratory (i.e., dioxin-like toxicity), including altered biochemical homeostasis, physical deformities, fetotoxicity, and teratogenesis. In addition to embryo mortality, PCBs cause edema and beak malformations often recognized as crossed beaks in double-crested cormorants (Firestone, 1973; Schrankel et al., 1982; Brunström and Darnerud, 1983: all as cited in Brunström, 1990).

Mammals – Mammals are susceptible to adverse effects from exposure to specific PCB congeners, including non-ortho and mono-ortho substituted PCBs, because their mechanism of action is similar to 2,3,7,8- TCDD (Leonards et al., 1995). Exposure to PCBs can cause mortality or serious reproductive complications in mammals. Other effects associated with PCB toxicity include anorexia, liver and kidney degeneration, and gastric ulcers, which have been observed in mink fed PCB-contaminated coho salmon (Wren, 1991).

7.2.4.2.3 DIOXINS AND FURANS

Polychlorinated dibenzo-*p*-dioxins (dioxins) and polychlorinated dibenzofurans (furans) are byproducts of several industrial reactions. The most significant sources of dioxins and furans appear to be their thermal formation during the incineration of municipal, industrial, and medical wastes (U.S. EPA, 1994). There are 75 individual dioxin congeners and 135 individual furan congeners with a range of levels of chlorination with 1 to 8 chlorines.

Benthic Invertebrates – Invertebrates appear to be relatively less sensitive to the effects of dioxin exposure than vertebrates such as fish, birds and mammals. However, reduced reproduction has been observed in snail and earthworm populations resulting from dioxin exposure (Eisler, 1986).

Fish – Early life stages of fish are more sensitive to mortality resulting from dioxin exposure. Species suffering from early life stage mortality include zebrafish, Japanese medaka, northern pike, rainbow trout, brook trout, and lake trout (Peterson and Walker, 1992). In salmonid sac fry, dioxin toxicity is characterized by pericardial and yolk-sac edema, subcutaneous hemorrhages, craniofacial malformations, and arrested growth and development (Spitsbergen et al., 1991; Walker et al., 1991).
Birds – Bird species in which dioxins and furans have been shown to cause embryo mortality include the chicken, ring-necked pheasant, turkey, eastern bluebird, mallard duck, domestic duck, golden-eye, herring gull, and black-headed gull (Brunstrom, 1988). Birds exhibit developmental toxicity in two ways (Peterson and Walker, 1992; Peterson et al. 1993). In the chicken embryo, pericardial, and subcutaneous edema, liver lesions, lymphoid toxicity, structural malformations, and mortality occur. In other bird species these embryotoxic effects are not seen; the only toxic effect that appears to be common to all bird species is embryo mortality (Carey et al., 1998).

Mammals – Tetrachlorodibenzo-*p*-dioxin can also adversely effect pregnancy maintenance, embryo or fetotoxicity, and postnatal development in mammals (Carey et al., 1998). Prenatal mortality has been observed in the mouse, rat, hamster, guinea pig, rabbit, mink, and rhesus monkey following dioxin exposure during pregnancy (Carey et al., 1998). Gestational exposure to dioxin produces a characteristic suite of responses that include, thymic hypoplasia, subcutaneous edema, and decreased fetal growth (Couture et al., 1990, Peterson et al., 1993).

7.2.4.2.4 CHLORINATED PESTICIDES

Chlordane, dieldrin, endrin, and heptachlor epoxide are members of the cyclodiene pesticides that are environmentally persistent and bioaccumulative chemicals that are stable in soil and resistant to photodegradation. As a result, they were used in greatest quantity as soil insecticides for the control of termites and soil-borne insects (Ware, 1997).

Gamma-BHC is a moderately toxic compound that is highly persistent in the environment. It is very stable in both freshwater and marine environments, and is resistant to photodegradation (EXTOXNET, 1996). It will disappear from water through secondary mechanisms such as adsorption to sediment, biological breakdown, and adsorption by aquatic animals through gills, skin, and food (EXTOXNET, 1996).

DDT and its metabolites are highly persistent in the environment, with reported half-lives of between 2 and 15 years in soil (ATSDR, 1992). Because of its persistence in soil, DDT can reach surface waters through erosion and atmospheric transport. The reported half-life for DDT in lake water is 56 days, while that in river water is 28 days. Field and laboratory studies have demonstrated very little breakdown of DDT in estuary sediments over the course of 46 days (EXTOXNET, 1996).

The mechanisms by which organochlorine pesticides cause ecotoxicity include narcosis (nonspecific toxicity) and more specific mechanisms that result in enhanced toxicity, such as respiratory uncouplers, acetylcholine esterase (AChE) inhibitors, and central nervous system convulants (Lipnick, 1993; McCarty and Mackay, 1993).

Benthic Invertebrates – Relatively little information was found on the toxicity of dieldrin to benthic invertebrates. The 5-hour median lethal concentration (LC_{50}) for brown shrimp was reported to range between 25 and 500 :g/L.

Heptachlor is highly toxic to freshwater invertebrates. LC_{50}s for freshwater invertebrates range from 0.9 :g/L for a 96-hour exposure with the stonefly, *Pteronarcella badia*, to 80 :g/L for a 48-hour exposure in the cladoceran, *Simnocephalus serrulatus* (U.S. EPA, 1980).

The only information found relating sediment concentrations of gamma-BHC to toxicity to benthic invertebrates was the freshwater TEL of 0.94 :g/L (NOAA, 1999). In the water column, gamma-BHC is highly toxic to aquatic invertebrates. Reported 96-hour LC_{50}s in aquatic invertebrates ranged from 4.5 :g/L in stoneflies to 460 :g/L in *Daphnia* sp. (EXTOXNET, 1996).
The toxicity of DDT-contaminated sediments to aquatic organisms has not been extensively studied. Spiked sediment bioassays using the freshwater amphipod *Hyalella azteca* found that the 10-day LC50s ranged from 11.0 to 49.7 mg/kg (TOC = 3.0 to 10.5 percent) (Nebeker, 1988).

In addition to direct toxicity, organochlorine pesticides are bioaccumulated in the tissues of benthic invertebrates. Trophic transfer and biomagnification of these compounds has been observed in a wide range of aquatic ecosystems (Carey et al., 1998).

Fish – Dieldrin is a relatively potent toxin in fish. The 96-hour LC_{50} for bluegill is 7.9 :g/L, while that in goldfish is 37 :g/L. In one study, exposure to 50 :g/L for five hours resulted in 100 percent mortality in mullet (Environmental Health Data Search, 1999).

Freshwater fish species are generally less sensitive to heptachlor than are invertebrate species. For freshwater fish species, 96-hour LC_{50}s ranged from 10.0 :g/L in rainbow trout to 320 :g/L in goldfish

(U.S. EPA, 1986). A 40-week chronic study was conducted using fathead minnows (*Pimephales promelas*) in which growth, reproduction, and survival were monitored. Concentrations of heptachlor tested were 1.84, 0.86, 0.43, 0.20, and 0.11 :g/L. All fish exposed to 1.84 :g/L were dead within 60 days. No adverse effects were reported in parental fish or their offspring at the other concentrations (U.S. EPA, 1980).

Gamma-BHC is also highly toxic to fish, with reported 96-hour LC_{50}s ranging from 1.7 to 90 :g/L in freshwater fish species.

Numerous acute toxicity studies demonstrate that DDT is acutely lethal to many aquatic organisms at low concentrations (Table 7-3). Additionally, the DDT metabolites DDD and DDE have also been shown to be acutely toxic to a number of fish species. Table 7-3 summarizes toxicity data for DDT, DDE, and DDD in a number of freshwater fish species.

Table 7-3. Acute Toxicity Data for DDT and Its Metabolites in Fish

Species	Concentration (:g/L)	Effect	Reference
DDT			
Fish (24 species)	0.6 – 180	LC_{50}	U.S. EPA 1980
Fish (19 species)	1.8 – 21.5	96-hour LC_{50}	Mayer & Ellersieck 1986
Fathead minnow	0.74	Chronic	U.S. EPA 1980
DDE			
Fish (3 species	32-240	96-hour LC_{50}	Mayer & Ellersieck 1986
DDD			
Fish (6 species)	14 – 4,400	96-hour LC_{50}	Mayer & Ellersieck 1986

In addition to its toxic effects, DDT bioaccumulates significantly in fish and other aquatic species, leading to long-term exposure. A half-life for elimination of DDT from rainbow trout was estimated to be 160 days (EXTOXNET, 1996). The reported bioconcentration factor for DDT ranges between 1,000 and 1,000,000 in various aquatic species (EXTOXNET, 1996). DDT and its metabolites biomagnify through the food web (ATSDR, 1992).

Birds – Birds are less sensitive to dieldrin than aquatic organisms. A medial lethal dose (LD_{50}) of 381 mg/kg body weight/day was reported in unspecified waterfowl (Environmental Health Data Search, 1999).

Heptachlor is moderately-to-highly toxic to birds. The reported acute oral LD_{50} in mallard ducks was 2,080 mg/kg, while the 5-day dietary LC_{50} in Japanese quail was 99 mg/kg. Other reported 8-day dietary LC_{50}s for heptachlor were 450 to 700 mg/kg in bobwhite quail, and 250 to 275 mg/kg in pheasant (EXTOXNET, 1996).

Gamma-BHC is slightly to moderately toxic to birds, with a reported LD_{50} of more than 2,000 mg/kg body wt/day in the mallard duck. The 5-day dietary LC_{50} of gamma BHC in Japanese quail was 490 mg/kg, while an LC_{50} of 561 mg/kg has been reported in pheasant. Eggshell thinning and reduced egg production has also occurred in birds exposed to gamma-BHC (EXTOXNET, 1996).

DDT may be slightly toxic to nearly non-toxic in birds. Reported dietary LD_{50}s ranged from greater than 2,240 mg/kg in mallard to 841 mg/kg in Japanese quail (EXTOXNET, 1996). There has been much concern over chronic exposure of bird species to DDT and effects on reproduction, especially eggshell thinning and embryo mortality. The mechanisms of eggshell thinning are not fully understood, although it is believed that predatory species of birds may be more sensitive to these effects. Laboratory studies on avian reproduction have demonstrated the potential for DDT and DDE to cause subtle changes in courtship behavior, delays in pairing and egg laying, and decreases in egg weight in ring doves and Bengalese finches (EXTOXNET, 1996).

Mammals – Some organochlorine pesticides such as o,p'-DDT, kepone and methoxychlor have estrogenic activity in wildlife. Many of these compounds, such as o,p'-DDT and kepone, have been shown to act by binding to the estrogen receptor. However, other organochlorine compounds can exert estrogenic or anti-estrogenic effects by other mechanisms (Carey et al., 1998). The overall impact of such estrogenic activity is disruption of normal reproductive functioning.

In addition, several chlorinated pesticides are known to affect mammalian immune system function. These pesticides include hexachlorobenzene, mirex, lindane, chlordane, dieldrin, and DDT and its metabolites (Carey, 1994). The immunotoxicity of these compounds has been demonstrated in several species and includes the loss of resistance to infections. In most cases, the mechanisms of action for these compounds are not well known.

7.2.4.2.5 TRACE ELEMENTS

Key factors that affect the form of sediment-associated trace metals present (speciation) include Eh (redox conditions), pH, porewater hardness, and the organic carbon content of the sediment. The redox conditions, pH, and the concentration of dissolved organic carbon in porewater influence the oxidation state and, thus, the dissolved concentration of the trace element. In this way, these factors provide some indication of the bioavailability of the metals present. Trace elements exhibit a range of binding affinities, with both organic and inorganic phases present in the sediment, resulting in varying concentrations of dissolved versus particulate metals. In addition, trace elements exhibit a range of stability constants with dissolved ligands, which determines the ratio of complexed to freely dissolved species in solution.

Total concentrations of trace elements in sediment are generally not predictive of their bioavailability. For certain metals, concentrations in porewater have been correlated with biological effects (DiToro et al., 1990). For several divalent metals, a key partitioning phase controlling cationic metal activity and toxicity in sediments appears to be acid-volatile sulfide (AVS) (DiToro et al., 1990, 1992; Carlson et al., 1991; Allen et al., 1993; Ankley et al. 1993). Simultaneously extracted metals (SEM) and AVS measurements can be made to assess the potential bioavailability of cadmium, copper, lead, nickel, and zinc.

The bioavailability of trace elements that form stable organo-metallic compounds is particularly complex. For example, methylmercury compounds are extremely toxic and are efficiently bioaccumulated through aquatic food chains (Wiener and Spry, 1996). Methylmercury is formed in aquatic sediments by microbial methylation of inorganic mercury (Eisler, 1987).

In freshwater, increasing water hardness decreases the toxicity of cadmium, chromium, copper, lead, nickel, silver, and zinc. The form of metal also effects toxicity. For example, methylmercury is more toxic than inorganic mercury. The combination of trace elements in the environment may result in additive, synergistic, or antagonistic effects, with the overall effect depending on the toxicity of the metals in question, the specific physical and chemical conditions of the site, and internal synergistic or antagonistic effects within organisms.

Benthic Invertebrates – Toxicity of trace elements to benthic organisms ranges widely, from slight reduction in growth rates to mortality. Oligochaetes and mollusks are generally less sensitive than other aquatic phyla (Leland and Kuwabara 1985). The most sensitive life stages of benthic organisms are generally the embryonic and larval stages.

Fish – Fish are exposed to trace elements both in the water column and through the consumption of contaminated prey organisms. Freshwater fish are generally more sensitive to the effects of trace elements than marine species, and the larval stages are generally most sensitive. Commonly observed effects include reductions in growth, survival, and fecundity.

Birds – Avian dietary toxicity studies have been conducted with a wide range of trace elements. The observed acute toxicity of the trace element can depend on the levels of metallothioneins in the bird. Ducks contained the highest levels of metallothioneins of a range of surveyed wildlife species (Brown et al., 1970, as cited in Eisler, 1985). Sublethal effects can include reproductive and behavioral modifications. Teratogenic effects have been documented in chicken embryos after eggs were injected with chromium (Ridgeway and Karnofsky, 1952; Gilani and Marano, 1979, as cited in Eisler, 1986). Similarly, the immersion of mallard eggs in solutions of methylmercury resulted in a significant incidence of skeletal embryonic malformations (Hoffman and Moore, 1979, as cited in Eisler, 1987).

Mammals – The only route of exposure for mammals that is being evaluated in this document is exposure through the aquatic food chain. Therefore, only trace elements that are known to be biomagnified through the food chain will be discussed. Methylmercury and lead are two trace elements that have been shown to be subject to trophic transfer and biomagnification. Organomercury compounds, especially methylmercury, are the most toxic mercury species for mammals. Larger mammals such as deer and seals appear to be more resistant to mercury than smaller mammals such as mink, cats, dogs, and river otters (Eisler, 1987). The reasons for these differences in sensitivity are unknown, but may be related to differences in metabolism and detoxification. Diet provides the major pathway for lead exposure. Food chain biomagnification of lead may be important for carnivorous marine mammals, such as the California sea lion and harbor seal (Eisler, 1988b).

7.2.4.3 RECEPTORS OF CONCERN

Biological studies indicate that a diverse aquatic community occupying several trophic levels is present in the lower Anacostia River. The selection of Receptors of Concern (ROCs) is based upon their potential presence in the study area, their sensitivity to contamination, and their potential for exposure to contaminants based on the identification of primary exposure pathways. The availability of appropriate toxicity information, exposure factors, and consumption data are also important considerations in the selection of ROCs.

Individual species have been selected for screening-level assessment to be representative ROCs for the fish and bird populations. These species were selected to be surrogate representatives of specific feeding strategies and of the potential transfer of specific classes of contaminants through the food chain.

Benthic invertebrates were evaluated as receptors of concern. These organisms are directly exposed to sediment contaminants. These organisms play a significant role in the functioning of riverine ecosystems as prey for larger animals, such as fish and crabs, and in the cycling of nutrients.

The largemouth bass was selected as a representative of a piscivorous fish species. The species is known to consume fish, crayfish, and insects (Scott and Crossman, 1973). Largemouth bass were not documented in the study area in the fish survey reported by Herson-Jones et al, (1994), but the species is known to reside in the watershed. Bass are more sensitive to habitat and water quality degradation than

many of the other freshwater bass and sunfishes (Scott and Crossman, 1973), so may avoid many reaches of the study area.

The brown bullhead was selected as a representative opportunistic benthic feeder. The species consumes a wide variety of benthic prey including mollusks, insects, crustaceans, worms, algae, fish, and fish eggs (Scott and Crossman, 1973). Brown bullheads were the most common freshwater resident observed in a fish survey conducted in the study area (Herson-Jones et al., 1994).

Birds and mammals associated with the study area may be exposed to contaminants through the consumption of forage fish and aquatic invertebrate species. The green heron and raccoon were selected as representative birds and mammals. The green heron consumes a variety of small nearshore fishes and invertebrates and has been documented in the Anacostia watershed. The raccoon is ubiquitous omnivore in the study area feeding on fish, aquatic and terrestrial invertebrates, and fruits.

7.2.5 EXPOSURE PATHWAYS

Potential exposure pathways are evaluated to determine which pathways are complete and important at the site. Identifying complete exposure pathways prior to a quantitative evaluation allows the assessment to focus on only those contaminants that can reach ecological receptors (U.S. EPA, 1997). An exposure pathway is considered complete if a contaminant can travel from a source to ecological receptors and can be taken up by the receptors via one or more exposure routes (U.S. EPA, 1997). Often many pathways are complete, but are of varying importance. It is therefore important to identify the key pathways that reflect maximum exposures within the ecosystem and constitute exposure pathways to ecological receptors sensitive to the contaminant (U.S. EPA, 1997). Pathways of exposure to PAHs, PCBs, dioxins and furans, pesticides, and trace elements for benthic invertebrates, fish, aquatic birds and mammals are relevant at this site. The exposure pathways for ROCs in the Anacostia River are illustrated in Figure 7-2. This assessment will not examine the sources of contaminants to the river and will focus on the risks associated with exposure to contaminants present in sediments and the water column throughout the lower river.

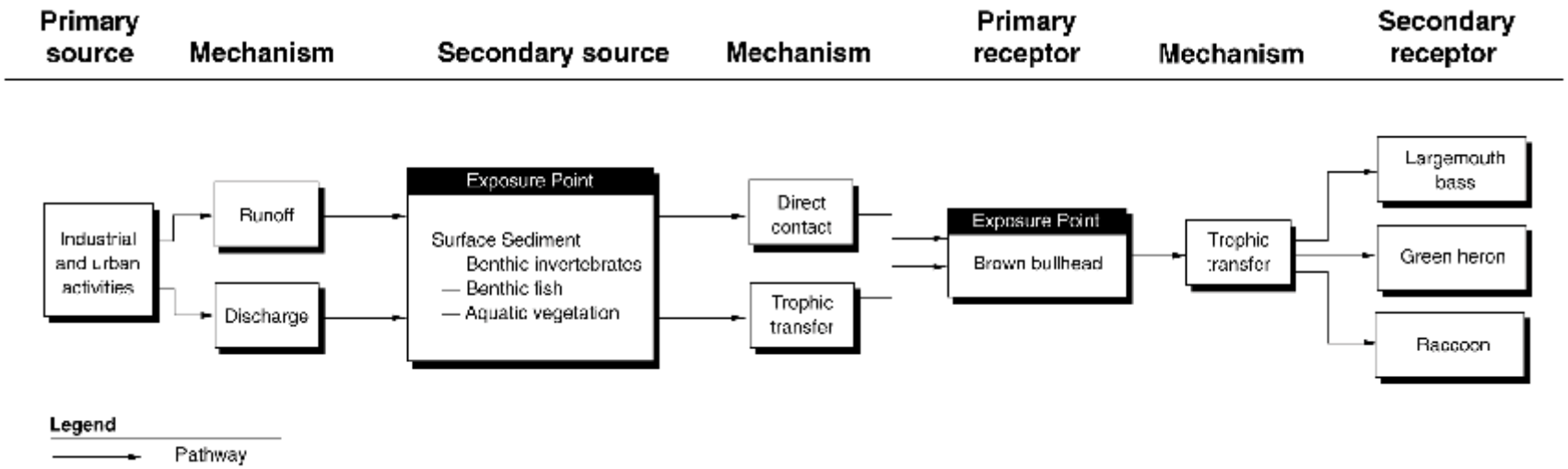


Figure 7-2. Potential exposure pathways for ecological receptors.

For benthic invertebrates, direct contact with water or sediment by the gills or integument are the primary exposure pathways (U.S. EPA, 1997). For lower-trophic-level fish, diet, and direct contact with water or sediment by the gills are the primary exposure pathways (U.S. EPA, 1997). For higher-trophic-level fish, diet can be an important exposure pathway for COPCs that are bioaccumulated or biomagnify, such as certain PCB congeners and methylmercury. The primary exposure pathway for birds and mammals is through the consumption of prey that have accumulated site-related contamination.

7.2.6 ASSESSMENT ENDPOINTS

As defined in U.S. EPA (1992), assessment endpoints are explicit expressions of the actual environmental values that are to be protected, such as ecological resources. Assessment endpoints are generally tied to the response of ecological receptor species to environmental stresses. Unless an ecological receptor is listed as a protected or endangered species, assessment endpoints are selected that are relevant to population-level rather than individual effects.

Assessment endpoints for the lower Anacostia River include:

X **Benthic invertebrates:** Assess the potential for adverse effects on diversity, abundance, growth, and survival of the benthic community

X **Fish:** Assess the potential for reproductive impairment and other adverse effects in both benthic fish and pelagic, predatory fish

X **Birds and mammals:** Assess the potential for adverse reproductive effects in aquatic birds and mammals

Measurement endpoints are measurable biological responses to the valued characteristics chosen as assessment endpoints; this definition is subject to change (U.S. EPA, 1997). The measurement endpoints corresponding to the assessment endpoints selected for this SLERA are presented below:

Benthic invertebrates:

X Sediment toxicity testing results compared to control sediment results

X Compare concentrations of COPCs in sediment to relevant screening concentrations

Fish:

X Compare surface water concentrations of COPCs to corresponding Ambient Water Quality Criteria (AWQC) values for the protection of aquatic biota

X Compare measured tissue concentrations of bioaccumulative COPCs in the brown bullhead as a representative benthic fish and largemouth bass as a predatory fish to literature values of toxic tissue residue levels

X Compare measured sediment concentrations of PAHs to relevant screening concentrations

Aquatic birds and mammals: Estimate concentrations of COPCs in prey, estimate dose to ROCs, and compare with literature toxicity values

7.3 BENTHIC INVERTEBRATES EVALUATION

The benthic invertebrates evaluation was conducted in two ways. First, the results of sediment toxicity testing that has been conducted using sediments collected within the Anacostia River are presented. Then, the maximum sediment contaminant concentrations are compared to corresponding benchmark values.

7.3.1 SCREENING LEVEL EXPOSURE ESTIMATE

7.3.1.1 SEDIMENT TOXICITY TESTING

Results of sediment toxicity testing conducted using sediments were available from two studies. The test organism, the amphipod *Hyalella azteca*, was the same in both studies however. Sediments were collected within Kenilworth Marsh (USFWS, 1997) and the Anacostia River (Velinsky et al., 1992) for bioassessment. In the Kenilworth Marsh study, toxicity testing was conducted with both bulk sediment samples and porewater samples. Significant toxicity was observed in three out of a total of nineteen tests conducted (USFWS, 1997). The Anacostia River sediments were tested as bulk sediment samples and significant toxicity was observed in four out of a total of thirty-two samples tested.

7.3.1.2 SEDIMENT CHEMISTRY SCREENING

Maximum sediment contaminant concentrations were selected from the sediment data sets compiled in the NOAA watershed database. The studies that were compiled in the database are presented in Appendix B. In conducting the screening evaluation the lower Anacostia was divided into three zones, the Upper River zone, the Lower River zone, and the Washington Ship Channel/Tidal Basin zone. The boundaries of these zones are discussed in Section 2.2 and illustrated in Figure 7-1.

7.3.2 SCREENING LEVEL EFFECTS ASSESSMENT

Sediment benchmark concentrations were selected from the literature to represent effects to benthic invertebrates. The selected sediment benchmark concentrations are presented in Table 7-4.

Table 7-4. Sediment benchmarks (:g/g dry weight)

	TEL
Trace elements	
Arsenic	10.8
Barium	0.7
Cadmium	0.6
Chromium, total	36.3
Copper	28
Lead	34.2
Manganese	615
Mercury	0.17
Nickel	19.5
Selenium	0.29
Silver	<0.5
Strontium	49
Vanadium	50
Zinc	94.2

PAHs	Benzo(a) anthracene	0.032
	Benzo(a)pyrene	0.032
	Chrysene	0.057
	Fluoranthene	0.11
	Phenanthrene	0.042
	Pyrene	0.053
	LPAHs	0.076
	HPAHs	0.193
	Total PAHs	0.264
PCBs	Aroclor 1254	0.032
	Aroclor 1260	0.032
	Total PCBs	0.032
Pesticides	Chlordane	0.0045
	Dieldrin	0.0029
	Heptaclor Epoxide	0.0006
	Lindane	0.0009
	DDD	0.00354
	DDE	0.0014
	total DDT	0.007
	Endrin	0.00267

Threshold effects levels (TELs) from the U.S. EPA ARCS program (U.S. EPA, 1996) were used as sediment benchmarks. TELs have been defined as the concentrations below which toxic effects are rarely observed (U.S. EPA, 1996). TELs were derived from freshwater exposures of *Hyalella azteca* using 28-day survival, growth, and reproductive endpoints (U.S. EPA, 1996). TELs were calculated as the geometric mean of the lower 15^{th} percentile concentrations of the effects data and the 50^{th} percentile of the no-effects data. In addition, TELs derived for freshwater sediments (Smith et al., 1996) were used for comparison for the pesticides because ARCS did not provide TELs for these contaminants. These TELs were calculated in the same manner as the ARCS values (U.S. EPA, 1996), but are based on a wider variety of bioassays and benthic community metrics. For several trace elements (barium, selenium, silver, and strontium) no TEL values were available, so background values were used (NOAA, 1999).

7.3.3 SCREENING LEVEL RISK CALCULATION

For screening-level risk calculations, the exposure estimates and the screening ecotoxicity values were combined using the hazard quotient approach to estimate risk (U.S. EPA, 1997). Hazard quotients (HQs) are defined as the ratio of the estimated dose or environmental exposure concentration at the site to a no observed effects level (NOAEL) for the contaminant. An HQ less than one indicates that that single contaminant alone is unlikely to cause the adverse biological effect reflected by the NOAEL (U.S. EPA, 1997).

The values for the upper river zone (Figure 7-1) are presented in Table 7-5. HQ values greater than one were calculated for all trace elements except arsenic and strontium. All maximum sediment PAH and pesticide concentrations were greater than the corresponding TEL values. Finally, the HQ calculated for the maximum total PCB concentration was fifty times greater then the total PCB TEL value.

Table 7-5. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Upper River

		TEL	Maximum Sediment Concentration (ppm)	$HQ_{(TEL)}$
Trace elements				
	Arsenic	10.8	6.46	0.60
	Barium	0.7	156.40	**223.43**
	Cadmium	0.6	2.62	**4.37**
	Chromium, total	36.3	134.00	**3.69**
	Copper	28	100.10	**3.58**
	Lead	34.2	224.00	**6.55**
	Manganese	615	643.10	**1.05**
	Mercury	0.17	0.59	**3.47**
	Nickel	19.5	50.51	**2.59**
	Selenium	0.29	nd	
	Silver	<0.5	nd	
	Strontium	49	21.72	0.44
	Vanadium	50	65.54	**1.31**
	Zinc	94.2	477.00	**5.06**
PAHs	Benzo(a) anthracene	0.032	0.78	**24.41**
	Benzo(a)pyrene	0.032	0.70	**21.72**
	Chrysene	0.057	1.10	**19.30**
	Fluoranthene	0.111	1.79	**16.13**
	Phenanthrene	0.042	0.85	**20.24**
	Pyrene	0.053	1.58	**29.81**
	LPAHs	0.076	1.68	**22.11**
	HPAHs	0.193	6.60	**34.20**
	Total PAHs	0.264	7.89	**29.89**
PCBs	Aroclor 1254	0.032	1.630	**50.94**
	Aroclor 1260	0.032	0.015	0.47
	Total PCBs	0.032	1.630	**50.94**
Pesticides	Chlordane	0.0045	0.196	**43.56**
	Dieldrin	0.0029	0.005	**1.72**
	Heptaclor Epoxide	0.0006	0.004	**6.67**
	Lindane	0.0009	0.002	**2.22**
	DDD	0.00354	0.082	**23.16**
	DDE	0.0014	0.047	**33.57**
	total DDT	0.007	0.149	**21.29**
	Endrin	0.00267	0.003	**1.12**

Table 7-6 presents the maximum sediment concentrations and calculated hazard quotients for the lower river zone. HQ values greater than one were calculated for all the trace elements, PAHs, total PCBs, and several of the chlorinated pesticides (dieldrin, DDD, DDE, total DDT, and endrin). The largest HQ values were calculated for the PAH compounds with HQ values ranging from 658–8,570.

Table 7-6. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Lower River

		TEL	Maximum Sediment Concentration (ppm)	$HQ_{(TEL)}$
Trace elements				
	Arsenic	10.8	26.90	**2.49**
	Barium	0.7	170.00	**242.86**
	Cadmium	0.6	3.18	**5.30**
	Chromium, total	36.3	155.50	**4.28**
	Copper	28	631.00	**22.54**
	Lead	34.2	775.00	**22.66**
	Manganese	615	800.00	**1.30**
	Mercury	0.17	2.70	**15.88**
	Nickel	19.5	69.70	**3.57**
	Selenium	0.29	1.10	**3.79**
	Silver	< 0.5	64.40	**128.80**
	Vanadium	50	68.10	**1.36**
	Zinc	94.2	512.00	**5.44**
PAHs	Benzo(a) anthracene	0.032	100.00	**3125.00**
	Benzo(a)pyrene	0.032	27.00	**843.75**
	Chrysene	0.057	86.00	**1508.77**
	Fluoranthene	0.111	110.00	**990.99**
	Phenanthrene	0.042	360.00	**8571.43**
	Pyrene	0.053	320.00	**6037.74**
	LPAHs	0.076	98.80	**1300.00**
	HPAHs	0.193	127.00	**658.03**
	Total PAHs	0.264	211.00	**799.24**
PCBs	Aroclor 1254	0.032	nd	
	Aroclor 1260	0.032	12.00	**375.00**
	Total PCBs	0.032	12.00	**375.00**
Pesticides	Chlordane	0.0045	0.0001	0.03
	Dieldrin	0.0029	0.0050	**1.72**
	Heptaclor Epoxide	0.0006	0.0003	0.53
	Lindane	0.0009	0.0001	0.11
	DDD	0.00354	0.17	**48.31**
	DDE	0.0014	0.07	**52.14**

	total DDT	0.007	0.32	**46.29**
	Endrin	0.00267	0.00	**1.28**

The Ship Channel and Tidal Basin sediments are evaluated in Table 7-7. HQ values greater than one were calculated for the trace elements, PAHs, total PCBs and all the organochlorine pesticides except endrin. The magnitude of the PAH HQ values were all greater than 100 and they were lower than those seen in the lower river with HQ values ranging from 155–396.

Table 7-7. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Ship Channel

		TEL	Maximum Sediment Concentration (ppm)	$HQ_{(TEL)}$
Trace elements				
	Arsenic	10.8	nd	
	Barium	0.7	nd	
	Cadmium	0.6	3.31	**5.52**
	Chromium, total	36.3	176.00	**4.85**
	Copper	28	348.00	**12.43**
	Lead	34.2	3630.00	**106.14**
	Manganese	615	nd	
	Mercury	0.17	9.22	**54.21**
	Nickel	19.5	nd	
	Selenium	0.29	nd	
	Silver	<0.5	nd	
	Vanadium	50	nd	
	Zinc	94.2	1090.00	**11.57**
PAHs	Benzo(a) anthracene	0.032	8.98	**280.63**
	Benzo(a)pyrene	0.032	6.48	**202.50**
	Chrysene	0.057	8.84	**155.09**
	Fluoranthene	0.111	19.71	**177.57**
	Phenanthrene	0.042	16.64	**396.19**
	Pyrene	0.053	14.61	**275.66**
	LPAHs	0.076	23.44	**308.42**
	HPAHs	0.193	65.98	**341.87**
	Total PAHs	0.264	89.41	**338.67**
PCBs	Aroclor 1254	0.032	nd	
	Aroclor 1260	0.032	nd	
	Total PCBs	0.032	3.3500	**104.69**
Pesticides	Chlordane	0.0045	0.1300	**28.89**
	Dieldrin	0.0029	0.0093	**3.21**
	Heptaclor Epoxide	0.0006	0.0028	**4.67**

Lindane	0.0009	0.0018	**2.00**
DDD	0.00354	0.1970	**55.65**
DDE	0.0014	0.1420	**101.43**
total DDT	0.007	0.8030	**114.71**
Endrin	0.00267	0.0015	0.56

7.4 FISH EVALUATION

The exposure of fish in the Anacostia River to bioaccumulative compounds such as dioxins and furans, PCBs, pesticides, trace elements, plus PAHs was evaluated using three approaches:

X Aqueous contaminant concentrations presented by Velinsky et al. (1999) were compared to their corresponding AWQC values.

X The exposures to bioaccumulative compounds (dioxins and furans, PCBs, pesticides, and trace elements) were assessed using a tissue residue effects approach. The concentrations of these compounds that accumulate in tissues are an integrative measure of all exposure pathways (i.e., dietary, respiratory, etc.). These tissue concentrations were compared to those known to cause injuries or those that have been associated with observations of adverse impacts.

X For PAHs, tissue effects concentrations are not relevant, as fish rapidly metabolize and excrete PAH compounds (Varanasi et al., 1989). Risks to fish from exposure to PAHs were evaluated by contrasting concentrations of the PAHs in sediments to sediment concentrations known to be injurious to fish or associated with observations of adverse effects.

In conducting the screening evaluation the lower Anacostia was divided into three zones, the Upper river zone, the Lower river zone, and the Washington Ship Channel/Tidal Basin zone. The boundaries of these zones are discussed in Section 7.2.2 and illustrated in Figure 7-1.

7.4.1 SCREENING LEVEL EXPOSURE ESTIMATE

7.4.1.1 WATER COLUMN SCREENING

Water column concentrations of a wide range of inorganic and organic contaminants from a recent study of the effects of stormwater runoff on water quality in Anacostia River were examined (Velinsky et al., 1999). This data was collected throughout 1998 and represents water quality both before and after major storm events. Water quality data will be screened using Ambient Water Quality Criteria (AWQC) for the protections of aquatic life (U.S. EPA, 1993). The AWQC values used in the screening are presented in Table 7-8.

Table 7-8. Chronic AWQC Values (:g/L)

	Criteria
INORGANICS	
NH4-N [(a)]	2,280
As(III)	150

Total As	150
Dissolved Cd	2.2 [b]
Dissolved Cr (VI)	11
Dissolved Cu	9 [b]
Dissolved Ni	52 [b]
Dissolved Pb	2.5 [b]
Dissolved Zn	120 [b]
ORGANICS	
otal PCBs	0.014
Heptachlor	0.0019
Aldrin	1.5 [c]
p,p'-DDE	1050 [c]
p,p'-DDT	0.0005

[a] chronic criterion as total ugN/L calculated based on pH=7.5 (EPA 1998)
[b] criteria calculated based on hardness of 100 mg/L CaCO3
[c] acute criteria used

7.4.1.2 TISSUE CONCENTRATION SCREENING

Tissue concentrations of contaminants in fish from the Anacostia have been measured in a range of studies that have been compiled into the NOAA Watershed Database. A listing of all the studies that have been used to develop the database is presented in Appendix B. Only whole body tissue concentrations for the two representative species, the brown bullhead and the largemouth bass, were evaluated. In addition, tissue concentrations reported for dead fish carcasses were not included due to potential changes in the fish tissue mass, as well as contaminant concentrations, following the death of the fish.

In the upper river zone, tissue concentrations of dioxins, PCBs, pesticides, and PAH were available for both the brown bullhead and the largemouth bass; concentrations of trace elements were only available for the brown bullhead. In the lower river zone, tissue concentrations of dioxins, PCBs, and pesticides were available for both the brown bullhead and the largemouth bass, only largemouth bass trace element tissue concentrations were available. In the Washington Ship Channel/Tidal Basin zone, only pesticide data was available for the largemouth bass.

There are no promulgated criteria for evaluating contaminant concentrations in fish tissue. However, concentrations associated with effects in toxicity tests and field studies are available and were reviewed. Lowest observed effects concentrations (LOECs) were selected from these studies to screen the estimated tissue concentrations against. Studies were identified from the U.S. Army Corps of Engineers (ACOE) ERED electronic database and from recent reviews of tissue residue effects data (Jarvinen and Ankley, 1999; Suter et al., 1999; Monosson, 1999; ESI, 1998).

7.4.1.2.1 POLYCHLORINATED BIPHENYLS

PCBs were marketed in the United States as mixtures of congeners known as Aroclors. Individual Aroclors contain different amounts of toxicologically important congeners. Therefore, it is important to know the identity of the individual Aroclors being summed to calculate a reported total PCB concentration. However, for the purposes of the screening-level risk evaluation, total PCB concentrations were screened. The type of PCB associated with each of the residue effects concentrations are identified in Table 7-9.

Selected LOECs for freshwater fish are presented in Table 7-9. The effects concentrations were selected as the lowest whole body concentrations associated with effects in freshwater species from two recent reviews (ESI, 1998; Suter et al., 1999). The results are consistent with the results of another review which estimated that whole body Aroclor 1254 concentrations of 5 :g/g wet weight or greater can result in reduced larval survival (Monosson, 1999). It should be noted that lower tissue concentrations associated with reproductive effects were reported for ovaries from baltic flounder (Von Westernhagen et al., 1981) and eggs from starry flounder, lake trout, and rainbow trout (Hogan and Brauhn, 1975; Hendricks et al., 1981; Spies et al., 1985; Mac and Edsall, 1991). In addition, increased mortality was seen in yearling coho salmon associated with PCB concentrations of 0.5 to 1.2 :g/g wet weight in the liver (Folmar et al., 1982). However, only whole-body concentrations were selected as screening concentrations.

Table 7-9. Lowest Observed Effects Concentrations of PCBs in Fish

Species	PCB Type	LOEC (:g/g wet wt)	Effects Endpoint	Reference
Lake Trout	Aroclor 1254	0.7[a]	fry mortality	Berlin et al. 1981 as cited in Suter et al. 1999
Atlantic salmon	Mixture of Aroclors 1016, 1221,1254,1260	3.0	reduced growth in alevins	Fisher et al. 1994
Adult fathead minnow	Aroclor 1254	13.7	reduced fecundity and frequency of reproduction	ACOE 1988
Fingerling channel catfish	Aroclor 1242	14.33	reduced growth	Hansen et al. 1976

[a] Geometric mean of the LOEC and NOEC

7.4.1.2.2 DIOXINS AND FURANS

Polychlorinated dibenzo-*p*-dioxins (dioxins) and polychlorinated dibenzofurans (furans) are byproducts of several industrial reactions. The most significant sources of dioxins and furans appear to be their thermal formation during the incineration of municipal, industrial, and medical wastes (U.S. EPA, 1994). There are 75 individual dioxin congeners and 135 individual furan congeners with a range of levels of chlorination with 1 to 8 chlorines.

Selected LOECs for freshwater fish are presented in Table 7-10. The effects concentrations associated with the lowest whole body concentrations associated with effects in freshwater species were obtained from a recent review of tissue effects concentrations (Jarvinen and Ankley, 1999).

Table 7-10. Lowest Observed Effects Concentrations of Dioxins and Furans

Species	Congener	LOEC (:g/g wet wt)	Effects Endpoint	Reference

Coho salmon	2,3,7,8-TCDD	0.125	survival	Miller et al., 1979
Rainbow trout	2,3,7,8-TCDD	0.00065-0.00258	growth	Branson et al., 1975
Rainbow trout	2,3,7,8-TCDD	0.00025	survival	Kleeman et al., 1986
Fathead minnow	2,3,7,8-TCDD	0.014	survival	Adams et al., 1986
Rainbow trout	2,3,7,8-TCDF	0.0093-0.0119	growth	Mehrle et al., 1988

7.4.1.2.3 PESTICIDES

There were fewer LOECs available for pesticides than for PCBs and mercury. Whole-body tissue concentrations associated with effects in freshwater fish species obtained from the ACOE ERED database were reviewed and the lowest reported LOEC or NOEC value was selected for each pesticide. The effects concentrations are presented in Table 7-11.

Table 7-11. Effects Concentrations of Pesticides in Fish

Contaminant	Species	Effects Concentration (:g/g ww)	Effects Endpoint	Reference
Dieldrin	Guppy	10.7	Reduced growth	Burnett and Liss 1990
Heptaclor Epoxide	Spot	0.016[a]	Mortality	Schimmel et al. 1976
Lindane	Fathead minnow	0.537[a]	Mortality	Macek et al. 1976
4,4'-DDD	Fathead minnow	0.6	Reproduction	Jarvinen et al. 1977
4,4'-DDE	Lake Trout	1.09	Mortality	Burdick et al. 1964
4,4'-DDT	Cutthroat Trout	0.567	Mortality	Cuerrier et al. 1967
	Brook Trout	0.89	Mortality	Macek 1968
	Lake Trout	2.93	Mortality	Burdick et al. 1964

[a]Value is an NOEC.

7.4.1.2.4 TRACE ELEMENTS

Tissue concentrations for the following trace elements were reported for the brown bullhead and the largemouth bass: arsenic, cadmium, lead, mercury, nickel, selenium, and zinc. In the following sections, the LOECs for whole body freshwater fish are presented. No effects concentrations for whole body freshwater fish were found for chromium or nickel.

Arsenic – The effect of temperature on the chronic toxicity of arsenate to rainbow trout has been studied (McGeachy and Dixon, 1990). Fish tested at 15° C were significantly less sensitive to the arsenate than fish tested at the same concentrations and lower temperatures (5° C). Both groups of fish had similar tissue residue concentrations and the greater sensitivity at lower temperatures might be

related to different toxicokinetics (Jarvinen and Ankley, 1999). In addition, juvenile fish appear to be more sensitive than adult fish to effects of arsenic species. The LOEC values for whole body freshwater fish are presented in Table 7-12.

Table 7-12. Lowest Observed Effects Concentrations of Arsenic in Fish

Species	Arsenic Species	Effects Concentration (:g/g wet wt)	Effects Endpoint	Reference
Rainbow Trout	Sodium arsenate	3.0	Reduced growth	McGeachy and Dixon 1990
Green sunfish	Sodium arsenate	6.7	mortality	Sorensen 1976
Bluegill	Sodium arsenite	2.24 - 11.7	Reduced growth and mortality	149

Cadmium – Cadmium is a surface active toxicant that causes mortality by disrupting ion-regulation by inhibiting ion-specific ATPases and opening tight junctions in the fish gill. This leads to a depletion of whole body ions as the higher internal concentrations of sodium and calcium ions diffuse into the surrounding water. Freshwater species appear to be more sensitive to the effects of cadmium than saltwater species. LOEC values for freshwater fish are presented in Table 7-13.

Table 7-13. Lowest Observed Effects Concentrations of Cadmium in Fish

Species	Effects Concentration (:g/g wet wt)	Effects Endpoint	Reference
Rainbow Trout	0.7 - 1.0	mortality	Pascoe et al., 1986
	0.96	growth	Kumada et al., 1973
	0.25	growth	Benoit et al., 1976
	0.14	survival	Hamilton et al., 1987a,b
Bluegill	0.35[a]	survival	Cearley and Coleman 1974
Bluegill		Reduced growth and mortality	

Lead – Holcombe et al. (1976) studied the effect of long-term lead exposure on three generations of brook trout. The tissue residue lead concentrations were measured in gill, liver, and kidney of the first- and second-generation adult fish. Whole body concentrations were reported for the third-generation embryos. The second- and third-generation fish exhibited acute and chronic effects at lower aqueous and tissue residue lead concentrations compared to the first generation fish. LOEC values from this study are summarized in Table 7-14.

Table 7-14. Lowest Observed Effects Concentrations of Lead in Fish

Species	Effects Concentration (:g/g wet wt)	Effects Endpoint	Reference
Brook trout[a]	0.40	mortality	Holcombe et al., 1976
	1.0-8.8	reduced growth	

[a] – third generation embryos
[b] – reduced survival measured in terms of hatchability

Mercury – Almost all of the studies reviewed were laboratory studies that dosed fish with either methylmercury or mercuric chloride in food or water. Uptake of methylmercury by aquatic organisms is both more rapid and more extensive than uptake of inorganic mercury (Biesinger et al., 1982 and others). LOECs for mercury in freshwater fish species are presented in Table 7-15. The selected concentrations represent the lowest whole-body mercury concentrations associated with effects from an extensive literature review (ESI, 1998). Lower effects concentrations were reported for the gonads of adult rainbow trout, associated with reduced larval survival in their offspring (Birge et al., 1979). For the purposes of this assessment, only whole-body tissue concentrations were selected as screening concentrations.

Table 7-15. Lowest Observed Effects Concentrations of Mercury in Fish

Species	Mercury Species	Effects Concentration (:g/g wet wt)	Effects Endpoint	Reference
Larval fathead minnow	Mercuric chloride	1.24	Reduced growth (length)	Snarski and Olson 1982
Larval fathead minnow	Mercuric chloride	1.36	Reduced growth (weight)	Snarski and Olson 1982
Rainbow trout	Methylmercury	1.8	Mortality	Hawryshyn and Mackay 1979
Juvenile walleye	Methylmercury	2.37	Reduced growth	Friedmann et al., 1996

Selenium – A recent review of effects associated with selenium tissue concentrations concluded that the toxicity of selenium depended both on the form of selenium and the route of exposure (i.e., aqueous vs. dietary) (Jarvinen and Ankley, 1999). LOEC concentrations for whole body freshwater fish are presented in Table 7-16.

Table 7-16. Lowest Observed Effects Concentrations of Selenium in Fish

Species	Selenium Species	Effects Concentration (:g/g wet wt)	Effects Endpoint	Reference
Rainbow Trout	Sodium selenite	1.9	mortality	Gissel-Nielsen and Gissel-Nielsen 1978
Chinook salmon[a]	Inorganic selenium	1.3	mortality	Hamilton et al. 1990
		0.66	Reduced growth	
Fathead minnow[a]	Sodium selenate	8.6	Reduced growth	Bennett et al. 1986
Fathead minnow	Selenate, selenite and seleno-L-methionine mixture	1.22	Reduced growth	Ogle and Knight 1989
Fathead minnow	Sodium selenate	9.5	Reduced growth	117
Bluegill	6:1 mixture of selenate to selenite	1.08	mortality	Cleveland et al. 1993

[a] - larvae

Zinc – Very few whole body zinc concentrations were found for freshwater fish species. The majority of values in a recently compiled database were either no-effect concentrations or organ concentrations (Jarvinen and Ankley, 1999). LOEC concentrations for whole-body freshwater fish are presented in Table 7-17.

Table 7-17. Lowest Observed Effects Concentrations of Zinc in Fish

Species	Effects Concentration (:g/g wet wt)	Effects Endpoint	Reference
Flagfish	44	mortality	Spehar 1976
	40	Reduced growth	

7.4.1.3 SEDIMENT THRESHOLD SCREENING OF PAHS

Pinkney et al. (2000) reported that 55 percent of brown bullheads collected from the Anacostia River had hepatic tumors. The prevalence of orocutaneous tumors was 23 percent. Baumann et al. (1996) reviewed the bullhead tumor survey data from the Great Lakes and stated that liver tumor rates greater than 9 percent and skin tumor rates greater than 20 percent were nearly always indicative of sites with contaminated sediments. Pinkney et al. (2000) reported a statistical association between PAH metabolite concentrations in fish bile and liver tumors, a further indication of a possible relationship between tumors and sediment PAH exposure. Correlations between sediment PAH concentrations and neoplasms in feral fish, and the induction of neoplasms in fish by exposure to contaminated sediment extracts, both support the hypothesis that some fish neoplasms and chronic responses result from exposure to PAHs present in the fish's environment (Black et al., 1980; Baumann et al., 1982; Baumann, 1984; Baumann et al., 1991; Horness et al., 1998).

A sediment quality threshold of 2 :g/g, derived from the relationship observed between sediment PAH concentrations and the prevalence of hepatic lesions in English sole (Horness et al., 1998), was selected for evaluating the potential exposure of fish to the PAHs in the sediments of the Anacostia River.

7.4.2 SCREENING-LEVEL RISK CALCULATIONS

7.4.2.1 SURFACE WATER SCREENING

The maximum surface water concentrations observed in this study are compared to the corresponding ambient water quality criteria in Tables 7-18 and 7-19. Only the upper river and lower river zones were evaluated as there was no corresponding water quality data for the Ship Channel from this study.

HQ values greater than one were calculated for lead and total PCBs in the upper river zone. In the lower river zone, none of the calculated HQ values were greater than one.

Table 7-18. Upper River Aqueous Contaminant Concentrations (Pinkney et al., 1993) and Corresponding AWQC Values (:g/L)

	Criteria	Max. Value	HQ
INORGANICS			
NH4-N [(a)]	2,280	371	0.16
As(III)	150	0.18	0.001
Total As	150	0.66	0.004
Dissolved Cd	2.2 [(b)]	0.041	0.02
Dissolved Cr (VI)	11	0.4	0.04
Dissolved Cu	9 [(b)]	4.02	0.45
Dissolved Ni	52 [(b)]	3.59	0.07
Dissolved Pb	2.5 [(b)]	3.32	**1.33**
Dissolved Zn	120 [(b)]	17.04	0.14
ORGANICS			
Total PCBs	0.014	0.017	**1.23**
Heptachlor	0.0019	0.000	0.02
Aldrin	1.5 [(c)]	nd	
p,p'-DDE	1050 [(c)]	0.001	0.000001
p,p'-DDT/IUPAC 176 [(d)]	0.0005	0.00032	0.64

[(a)] chronic criterion as total :gN/L calculated based on pH=7.5 (EPA 1998)
[(b)] criteria calculated based on hardness of 100 mg/L CaCO3
[(c)] acute criteria used
[(d)] co-elution of p,p'-DDT and PCB congener IUPAC #176

Table 7-19. Lower River Aqueous Concentrations (Pinkney et al., 1993) and Corresponding AWQC Values (:g/L)

	Criteria	Max. Value	HQ
INORGANICS			
NH4-N [(a)]	2,280	902	0.40
As(III)	150	0.17	0.001
Total As	150	0.59	0.004
Dissolved Cd	2.2 [(b)]	0.3	0.14
Dissolved Cr (VI)	11	3.73	0.34
Dissolved Cu	9 [(b)]	2.35	0.26
Dissolved Ni	52 [(b)]	1.94	0.04
Dissolved Pb	2.5 [(b)]	1.26	0.50
Dissolved Zn	120 [(b)]	15.08	0.13
ORGANICS			
Total PCBs	0.014	0.0094	0.67
Heptachlor	0.0019	0.0003	0.16
Aldrin	1.5 [(c)]	0.00012	0.0001

p,p'-DDE	1050 [c]	0.00068	0.000001
p,p'-DDT/IUPAC 176 [d]	0.0005	0.00065	1.30

[a] chronic criterion as total ugN/L calculated based on pH=7.5 (EPA 1998)
[b] criteria calculated based on hardness of 100 mg/L CaCO3
[c] acute criteria
[d] co-elution of p,p'-DDT and PCB congener IUPAC #176

7.4.2.2 TISSUE EFFECT CONCENTRATIONS

Risk were estimated as HQs, calculated using the lowest of the LOECs for PCBs, dioxins and furans, pesticides, and trace elements from Tables 7-9 through 7-17. When the LOEC value was based on a mortality endpoint, a screening value was calculated as the LOEC/10 in order to estimate the chronic LOEC value from the acute value.

The maximum tissue concentrations and corresponding LOEC values for the upper river zone are presented in Table 7-20. The only HQ greater than one for the brown bullhead was the value for lead (HQ equal to 2.59). The only HQ greater than one calculated for the largemouth bass was for total PCBs (HQ equal to 1.39).

Table 7-20. Upper River Maximum Fish Tissue Concentrations Compared to Corresponding LOEC

	LOEC (ug/g wet wt)	Upper River Brown Bullhead	HQ	Largemouth bass	HQ
Dioxins and Furans:					
2,3,7,8-TCDD	0.000025 [a]	0.0000002	0.0088	0.0000005	0.0180
2,3,7,8-TCDF	0.0093	0.0000007	0.0001	0.0000006	0.0001
PCBs:					
Total PCB	0.7	0.29300	0.42	0.97	**1.39**
Trace Elements:					
Arsenic	2.24	0.034	0.02	NA	
Cadmium	0.014 [a]	0.006	0.40	NA	
Lead	0.04 [a]	0.104	**2.59**	NA	
Mercury	1.24	0.051	0.04	NA	
Selenium	0.66	0.193	0.29	NA	
Zinc	40	5.920	0.15	NA	
Pesticides:					

Dieldrin	10.7	0.009	0.001	0.020	0.002
Heptaclor Epoxide	0.016	0.0001	0.01	0.001	0.056
Lindane	0.537	0.0003	0.001	NA	
DDD	0.6	0.040	0.07	0.120	0.200
DDE	0.109 [(a)]	0.049	0.45	0.100	0.917
DDT	0.0567 [(a)]	0.001	0.03	0.001	0.009

[(a)] Original LOEC was based on mortality endpoint; screening value = LOEC/10

The calculated HQ values for the lower river zone are presented in Table 7-21. HQ values greater than one were calculated for total PCB concentrations in both the brown bullhead and the largemouth bass (HQs equal to 1.82 and 1.36, respectively). In addition, the HQ calculated for lead in the largemouth bass was 1.08. Finally, the HQ values calculated for the pesticides, DDD and DDE, were greater than one for both the brown bullhead and the largemouth bass.

Table 7-21. Lower River Maximum Fish Tissue Concentrations Compared to Corresponding LOEC

	LOEC (ug/g wet wt)	Lower River Brown Bullhead	HQ	Largemouth bass	HQ
Dioxins and Furans:					
2,3,7,8-TCDD	0.000025 (a)	0.00001	0.31	0.00000322	0.13
2,3,7,8-TCDF	0.0093	0.0000024	0.0003	0.0000004	0.00004
PCBs:					
Total PCB	0.7	1.27400	**1.82**	0.954	**1.36**
Trace Elements:					
Arsenic	2.24	NA		0.099	0.04
Cadmium	0.014 (a)	NA		0.004	0.30
Lead	0.04 (a)	NA		0.043	**1.08**
Mercury	1.24	NA		0.078	0.06
Selenium	0.66	NA		0.274	0.42
Zinc	40	NA		4.559	0.11
Pesticides:	10.7				
Dieldrin	0.016	0.00025	0.0156	0.020	**1.25**
Heptaclor Epoxide	0.537	0.01700	0.03	0.005	0.01
Lindane	0.6	0.00120	0.002	0.000	0.0003
DDD	0.109 (a)	0.14000	**1.28**	0.130	**1.19**
DDE	0.0567 (a)	0.13000	**2.29**	0.150	**2.65**
DDT	0.567	0.01000	0.02	0.004	0.01

(a) Original LOEC was based on mortality endpoint; screening value = LOEC/10

The only tissue data available for the Ship Channel and Tidal Basin were PCB and pesticide data for largemouth bass (Table 7-22). HQ values greater than one were calculated for total PCB, dieldrin, DDD, and DDE. The largest HQ value was calculated for DDE (HQ equal to 11.1).

Table 7-22. Ship Channel Maximum Fish Tissue Concentrations Compared to the Corresponding LOEC

	LOEC (ug/g wet wt)	Ship Channel Brown Bullhead	HQ	Largemouth bass	HQ
Dioxins and Furans:					
2,3,7,8-TCDD	0.000025 [(a)]	NA		NA	
2,3,7,8-TCDF	0.0093	NA		NA	
PCBs:					
Total PCB	0.7	NA		4.7	**6.71**
Trace Elements:					
Arsenic	2.24	NA		NA	
Cadmium	0.014 [(a)]	NA		NA	
Lead	0.04 [(a)]	NA		NA	
Mercury	1.24	NA		NA	
Selenium	0.66	NA		NA	
Zinc	40	NA		NA	
Pesticides:	10.7				
Dieldrin	0.016	NA		0.04	**2.500**
Heptaclor Epoxide	0.537	NA		NA	
Lindane	0.6	NA		NA	
DDD	0.109 [(a)]	NA		0.36	**3.30**
DDE	0.0567 [(a)]	NA		0.63	**11.11**
DDT	0.567	NA		0.06	0.11

[(a)] Original LOEC was based on mortality endpoint; screening value = LOEC/10

7.4.2.3 RESULTS OF SEDIMENT THRESHOLD SCREENING OF PAHS

The maximum sediment total PAH concentration in each area was compared to the sediment quality threshold of 2 :g/g. The resulting HQ for the upper river zone was 3.9. The HQ values calculated for the lower river and the Ship Channel sediments were considerably higher (HQs of 105 and 45, respectively).

7.5 AQUATIC BIRD EVALUATION

In conducting the screening evaluation for aquatic birds, the lower Anacostia was divided into three zones, the Upper river zone, the Lower river zone, and the Washington Ship Channel/Tidal Basin zone. The boundaries of these areas are discussed in Section 2.2 and illustrated in Figure 7-1.

7.5.1 SCREENING LEVEL EXPOSURE ESTIMATE

Exposure of avian receptors to COPCs associated with Anacostia River sediments was estimated based on the assumption that contaminant exposure was entirely through diet. The selected bird receptor is the green heron which consumes a mixed diet of fish, invertebrates, and various insects. Dietary composition for the green heron was reported to have a lower fraction of fish than some reported diets for the larger great blue heron. In order to assure a conservative exposure estimate, the dietary composition reported for the larger great blue heron (U.S. EPA, 1993) was used for the green heron (Table 7-23).

Table 7-23. Dietary Composition for Green Heron (U.S. EPA, 1993)

Species	Percent fish	Percent Invertebrates	Percent incidental sediment ingestion
Green Heron[1]	94	6	1.8[2]

[1] – composition data for the great blue heron from USEPA (1993) used for green heron

[2] – calculated from sediment ingestion rate for sandpiper of 30 percent multiplied by the percent invertebrates consumed by the heron

The maximum measured fish tissue contaminant concentrations was used to calculate the dose resulting from the fish portion of the diet of the raccoon and the green heron. No invertebrate tissue concentrations are available so the invertebrate tissue concentrations were estimated from the maximum sediment concentrations using biota to sediment accumulation factors (BSAFs). For organic contaminants such as PCBs and pesticides, BSAFs are calculated by dividing the lipid-normalized concentrations of a chemical in an organism by the organic-carbon-normalized (OC-normalized) concentrations of the same chemical in sediment (Equation 1):

$$\text{BSAF} = \frac{\text{lipid-normalized tissue concentration}}{\text{OC-normalized sediment concentration}} \qquad \textbf{Eq. 7-1}$$

An estimated tissue concentration can thus be calculated by multiplying the BSAF by the OC-normalized sediment concentration (Equation 2):

$$\text{Estimated lipid-normalized tissue concentration} = \text{BSAF} \times \text{OC-normalized sediment concentration} \qquad \textbf{Eq. 7-2}$$

BSAFs used to estimate tissue concentrations of PCBs and pesticides are presented in Table 7-24. BSAFs for PCBs and pesticides represent median BSAFs for benthic organisms, including benthically-associated fish (spot, channel catfish, croaker white perch, fathead minnow, and scup), calculated by Tracey and Hansen (1995). An extensive analysis of differences in BSAFs for individual species and trophic levels concluded that the calculated BSAFs were similar for various species both within the same trophic level and between different trophic levels (Tracey and Hansen, 1995). However, the use of a single BSAF for an entire class of contaminants, such as PCBs or pesticides, is a simplification of the complexity of the chemistry of the individual compounds.

Table 7-24. BSAF Values for PCBs and Pesticides

Contaminant	BSAF	Reference
PCBs	1.64	Tracey and Hansen (1995)
Pesticides	1.96	Tracey and Hansen (1995)

A biota-to-sediment ratio of 1 was used to estimate the invertebrate trace element tissue concentrations. This value appears to be conservative for mercury based on the sediment and tissue concentrations presented by Suter et al. (1999) for largemouth bass, bluegill, gizzard shad, and channel catfish collected from a large river-reservoir system.

Invertebrate tissue concentrations of PCBs, trace elements, and pesticides in the upper river, the lower river, and the Ship Channel zones were estimated, using these values, and are presented in Tables 7-24 through 7-27.

Table 7-25. Estimated Maximum Invertebrate Tissue Concentrations in the Upper River

		Maximum sediment conc (ppm dry wt)	C_{org} TOC normalized	BSAF	Tissue conc dry wt		Tissue conc wet weight [a]
Trace elements							
	Arsenic	6.46		1	6.46		1.292
	Cadmium	2.62		1	2.62		0.524
	Lead	224.00		1	224		44.800
	Mercury	0.59		1	0.59		0.118
	Selenium	nd		1			
	Zinc	477.00		1	447		89.400
PCBs	Aroclor 1254	1.630	45.79	1.64	3.75	[b]	0.751
	Aroclor 1260	0.015	0.42	1.64	0.03	[b]	0.007
	Total PCBs	1.630	45.79	1.64	3.75	[b]	0.751
							0.000
Pesticides	Chlordane	0.196	5.51	1.96	0.54	[b]	0.108
	Dieldrin	0.005	0.14	1.96	0.01	[b]	0.003

	Heptaclor Epoxide	0.004	0.11	1.96	0.01 [b]	0.002
	Lindane	0.002	0.06	1.96	0.01 [b]	0.001
	DDD	0.082	2.30	1.96	0.23 [b]	0.045
	DDE	0.047	1.32	1.96	0.13 [b]	0.026
	total DDT	0.149	4.19	1.96	0.41 [b]	0.082
	Endrin	0.003	0.08	1.96	0.01 [b]	0.002

[a] calculated assuming 80 percent moisture (Stephan et al., 1985)
[b] calculated assuming 5 percent lipid and using area average TOC of 3.56 percent

Table 7-26. Estimated Maximum Invertebrate Tissue Concentrations in the Lower River

		Maximum sediment conc (ppm dry wt)	C_{org} TOC normalized	BSAF	Tissue conc dry wt	Tissue conc wet weight [a]
Trace elements						
	Arsenic	26.90		1	26.9	5.380
	Cadmium	3.18		1	3.18	0.636
	Lead	775.00		1	775	155.000
	Mercury	2.70		1	2.7	0.540
	Selenium	1.10		1	1.1	0.220
	Zinc	512.00		1	512	102.400
PCBs	Aroclor 1254	nd		1.64		
	Aroclor 1260	12.00	353.98	1.64	29.03 [b]	5.805
	Total PCBs	12.00	353.98	1.64	29.03 [b]	5.805
Pesticides	Chlordane	0.0045	0.13	1.96	0.01 [b]	0.003
	Dieldrin	0.0029	0.09	1.96	0.01 [b]	0.002
	Heptaclor Epoxide	0.0006	0.02	1.96	0.00 [b]	0.00035
	Lindane	0.0009	0.03	1.96	0.00 [b]	0.001
	DDD	0.00354	0.10	1.96	0.01 [b]	0.002
	DDE	0.0014	0.04	1.96	0.00 [b]	0.001
	total DDT	0.007	0.21	1.96	0.02 [b]	0.004
	Endrin	0.00267	0.08	1.96	0.01 [b]	0.002

[a] calculated assuming 80 percent moisture (Stephan et al., 1985)
[b] calculated assuming 5 percent lipid and using area average TOC of 3.39 percent

Table 7-27. Estimated Maximum Invertebrate Tissue Concentrations in the Ship Channel

		Maximum sediment conc (ppm dry wt)	C_{org} TOC normalized	BSAF	Tissue conc dry wt	Tissue conc wet weight [a]
Trace elements						
	Arsenic	nd		1	nd	
	Cadmium	3.31		1	3.31	0.662
	Lead	3630.00		1	3630.00	726.000
	Mercury	9.22		1	9.22	1.843
	Selenium	nd		1	nd	
	Zinc	1090.00		1	1090.00	218.000
PCBs	Aroclor 1254	nd				
	Aroclor 1260	nd				
	Total PCBs	3.3500	87.01	1.64	7.14 [b]	1.427
						0.000
Pesticides	Chlordane	0.1300	3.38	1.96	0.33 [b]	0.066
	Dieldrin	0.0093	0.24	1.96	0.02 [b]	0.005
	Heptaclor Epoxide	0.0028	0.07	1.96	0.01 [b]	0.001
	Lindane	0.0018	0.05	1.96	0.00 [b]	0.001
	DDD	0.1970	5.12	1.96	0.50 [b]	0.100
	DDE	0.1420	3.69	1.96	0.36 [b]	0.072
	total DDT	0.8030	20.86	1.96	2.04 [b]	0.409
	Endrin	0.0015	0.04	1.96	0.00 [b]	0.001

[a] calculated assuming 80 percent moisture (Stephan et al., 1985)
[b] calculated assuming 5 percent lipid and using area average TOC of 3.85 percent

Food ingestion rates for the green heron and raccoon were estimated using the procedures outlined in U.S. EPA's *Wildlife Exposure Factors Handbook* (1993). The rate of food consumption that an animal must achieve to meet its metabolic needs can be calculated by dividing its free-living (or field) metabolic rate (FMR) by the metabolizable energy in its food. Metabolizable energy (ME) is the gross energy in a unit of food consumed minus the energy lost in urine and feces (U.S. EPA, 1993).

The generic equation for estimating oral doses of contaminants in food for wildlife species is:

$$ADD_{pot} = \sum_{k=1}^{m} (C \times FR_k \times NIR_k) \qquad \textbf{Eq. 7-3}$$

where:

ADD_{pot} = potential average daily dose (e.g., mg/kg/day)

C_k = average contaminant concentration in food type k (e.g., mg/kg wet weight)

FR_k = fraction of intake of food type k that is contaminated (unitless). For example, if k in an animal's diet were salmon, FR_k for salmon would equal the fraction of the salmon consumed that is contaminated at level C_k. If all of the salmon consumed were contaminated at level C_k, then FR_k would equal one.

NIR_k = Normalized ingestion rate of food type k on a wet-weight basis (e.g., in g/g-day)

m = number of contaminated food types

Estimation of contaminant dose for heron in the Anacostia River is based only on consumption of benthic invertebrates and fish with incidental sediment ingestion, and it is assumed that 100 percent of the prey items are contaminated, so that FR_k is equal to 1. Therefore, the only item left to calculate is the normalized ingestion rate.

The normalized ingestion rate can be calculated as follows:

$$NIR = \frac{FMR \times ME_{avg}}{BW} \qquad \textbf{Eq. 7-4}$$

where:

NIR = normalized ingestion rate (g/g BW/day)
BW = body weight in grams
FMR = estimated field metabolic rate (kcal/day)
ME_{avg} = estimated average metabolizable energy of diet (kcal/g wet wt)

FMRs have been calculated for a number of animal species, including birds and mammals. The FMR for non-passerine birds, as presented in U.S. EPA (1993), is:

$$FMR_{(kcal/day)} = 1.146(BW)^{0.749}$$

Eq. 7-5

Mean body weight is 212 g for the green heron. For this risk assessment the body weight was calculated by taking the average of the mean male and female body weights.

The metabolizable energy (ME) of a fish diet as presented in U.S. EPA (1993) is 0.95 kcal/g ww for birds. The estimated normalized ingestion rates for the green heron is 0.28g/g body weight/day.

7.5.2 DIOXINS AND FURANS TEQ EVALUATION

Although individual dioxin and furan congeners have similar modes of toxic action, they all have differing degrees of potency due to their specific stereochemistry. Toxicity Equivalency Factors (TEFs) have been developed to express the toxicity of individual dioxin and furan concentrations in terms of the equivalent toxicity of a particular benchmark concentration, that of 2,3,7,8-TCDD. A number of TEF values have been developed to calculate 2,3,7,8-TCDD equivalent concentrations (TEQ) for birds. The WHO TEF values presented by Ahlborg et al. (1994) were used to calculate TEQ values for the green heron. The maximum fish tissue concentration reported for either the largemouth bass or the brown bullhead for each dioxin and furan congener was selected for use in this screening evaluation. The congener concentrations, TEF values and calculated TEQ values for the upper river and lower river zones are presented in Tables 7-28 and 7-29, respectively.

Table 7-28. WHO TEF Values for Birds and Calculated TEQ Values Based on the Maximum Fish Tissue Concentrations for the Upper River

	Congeners	Bird WHO TEF	Max. fish conc. ng/kg	Bird TEQ
Dioxins				
	2,3,7,8-TCDD	1	0.45	0.45
	1,2,3,78-PeCDD	1	0.55	0.55
	1,2,3,4,7,8-HxCDD	0.05	1.05	0.0525
	1,2,3,6,7,8-HxCDD	0.01	na	
	1,2,3,7,8,9-HxCDD	0.1	na	
	1,2,3,4,6,7,8-HpCDD	0.001	0.8	0.0008
	OCDD	na	na	
Furans				
	2,3,7,8-TCDF	1	0.7	0.7
	1,2,3,7,8-PeCDF	0.1	5	0.5
	2,3,4,7,8-PeCDF	1	4.75	4.75
	1,2,3,4,7,8-HxCDF	0.1	0.6	0.06
	1,2,3,6,7,8-HxCDF	0.1	na	
	1,2,3,7,8,9-HxCDF	0.1	na	
	2,3,4,6,7,8-HxCDF	0.1	0.95	0.095
	1,2,3,4,6,7,8-HpCDF	0.01	13.5	0.135
	1,2,3,4,7,8,9-HpCDF	0.01	na	
	OCDF	0.0001	31.5	0.00315

			Sum TEQ	7.29645

Table 7-29. WHO TEF Values for Birds and Calculated TEQ Values Based on the Maximum Fish Tissue Concentrations for the Lower River

	Congeners	Bird WHO TEF	Max. fish conc. ng/kg	Bird TEQ
Dioxins				
	2,3,7,8-TCDD	1	2.8	2.8
	1,2,3,78-PeCDD	1	3.9	3.9
	1,2,3,4,7,8-HxCDD	0.05	1.5	0.075
	1,2,3,6,7,8-HxCDD	0.01	4.9	0.049
	1,2,3,7,8,9-HxCDD	0.1	na	
	1,2,3,4,6,7,8-HpCDD	0.001	6.2	0.0062
	OCDD	na	57.1	
Furans				
	2,3,7,8-TCDF	1	2.4	2.4
	1,2,3,7,8-PeCDF	0.1	3	0.3
	2,3,4,7,8-PeCDF	1	4.6	4.6
	1,2,3,4,7,8-HxCDF	0.1	2.5	0.25
	1,2,3,6,7,8-HxCDF	0.1	1.6	0.16
	1,2,3,7,8,9-HxCDF	0.1	3.6	0.36
	2,3,4,6,7,8-HxCDF	0.1	0.2	0.02
	1,2,3,4,6,7,8-HpCDF	0.01	1.85	0.0185
	1,2,3,4,7,8,9-HpCDF	0.01	2.55	0.0255
	OCDF	0.0001	17.7	0.00177
			Sum TEQ	14.96597

The TRVs used to evaluate the green heron TEQ dose were derived from feeding studies conducted by Summer et al. (1996a,b). The studies were conducted with Babcock white leghorn chickens which were fed carp from Saginaw Bay at three treatment levels. The high dose treatment resulted in reduced hatchability of eggs and an increase in the overall deformity rate in embryos and chicks during weeks 1 through 10 compared with controls.

The TEQ dose, TRV values, and calculated HQ values for the green heron in both the upper river and lower river zones are presented in Table 7-30. HQs greater than one were calculated for both the raccoon and the green heron in both the upper and lower river zones using the NOAEL TEQ value as the TRV. The largest HQ value (HQ: 12.2) was calculated for the raccoon in the lower river. When the LOAEL TEQ values were used as TRV values, all the resulting HQ values were less than one.

Table 7-30. Comparison to 2,3,7,8-TCDD TEQs to NOAEL Doses

		TEQ	Ingestion Dose	NOAEL TEQ	**HQ**	LOAEL TEQ	**HQ**
Upper River							
	Green Heron	7.30	0.28 2.04	1.05	**1.95**	9.94	0.21
Lower River							
	Green Heron	14.97	0.28 4.19	1.05	**3.99**	9.94	0.42

7.5.3 SCREENING LEVEL EFFECTS ASSESSMENT

The estimated wildlife NOAELs for great blue heron reported by Sample et al. (1996) were used as TRV values for the green heron. The TRV values and the calculated dose for both species are presented in Tables 7-31 through 7-33 for the upper river zone, the lower river zone, and the Ship Channel and Tidal Basin. The speciation of mercury in the fish tissues was not reported. Therefore, the dose of mercury to each species was evaluated twice, first assuming that the concentration was entirely inorganic mercury, then assuming the concentration was entirely methyl mercury.

In the upper river zone, green heron HQs greater than one were calculated for total DDT and methyl mercury (Table 7-31). The largest HQ was calculated for methyl mercury in the green heron (HQ = 26.4).

Table 7-31. Green Heron Dose Compared to TRV Values for the Upper River

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww [(a)]	Dose (:g/gbw/d) [(b)]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.0000005	na	na	0.0000001	0.000014	0.01
2378-TCDF	0.0000007	na	na	0.0000002	0.000001	0.18
PCBs						
Total PCB	0.97	0.751	0.2445	0.269	0.41	0.66
Pesticides						
Dieldrin	0.0095	0.003	0.00075	0.003	0.077	0.03
Heptachlor Epoxide	0.0001	0.002	0.0006	0.0001	na	
Lindane	0.00031	0.001	0.0003	0.0001	2	0.00005
Total DDT	0.0901	0.082	0.022	0.025	0.003	**8.40**
Trace Elements						
Arsenic	0.034	1.292	0.969	0.036	2.5	0.01
Cadmium	0.0057	0.524	0.393	0.012	1.45	0.01
Lead	0.104	44.8	33.6	0.949	1.13	0.84
Mercury	0.051	0.118	0.0885	0.016	0.45	0.04
Methyl mercury	0.051	0.118	0.0885	0.016	0.0006	**26.42**

Selenium	0.193	nd	nd	0.051	0.5	0.10
Zinc	5.92	89.4	71.55	3.421	14.5	0.24

(a) calculated from dry weight concentrations assuming 85 percent moisture

(b) based on diet of 94 percent fish, 6 percent invertebrates, and 1.8 percent incidental sediment ingestion, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day

In the lower river zone, HQs greater than one were calculated for total PCBs, total DDT, lead, and methyl mercury in the green heron (Table 7-32). The largest HQs were calculated for total DDT and methyl mercury (HQs of 24.6 and 40.9, respectively).

Table 7-32. Green Heron Dose Compared to TRV Calues for the Lower River

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww (a)	Dose (:g/gbw/d) (b)	TRV	HQ
Dioxins and furans						
2378-TCDD	0.00001	na	na	0.000003	0.000014	0.19
2378-TCDF	0.0000024	na	na	0.000001	0.000001	0.63
PCBs						
Total PCB	1.27	5.805	1.8	0.441	0.41	**1.08**
Pesticides						
Dieldrin	0.02	0.002	0.000675	0.005	0.077	0.07
Heptachlor Epoxide	0.017	0.00035	0.00009	0.004	na	
Lindane	0.0012	0.001	0.000135	0.0003	2	0.0002
Total DDT	0.28	0.004	0.001	0.074	0.003	**24.59**
Trace Elements						
Arsenic	0.099	5.38	4.04	0.137	2.5	0.05
Cadmium	0.0041	0.636	0.477	0.014	1.45	0.01
Lead	0.043	155	116	3.200	1.13	**2.83**
Mercury	0.078	0.54	0.405	0.032	0.45	0.07
Methyl mercury	0.078	0.54	0.405	0.026	0.0006	**43.78**
Selenium	0.27	0.22	0.165	1.792	0.5	**3.58**
Zinc	4.56	102.4	76.8	1.587	14.5	0.11

(a) calculated from dry weight concentrations assuming 85 percent moisture

(b) based on diet of 94 percent fish, 6 percent invertebrates, and 1.8 percent incidental sediment ingestion, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day

Finally, in the Washington Ship Channel/Tidal Basin zone, HQs greater than one were calculated for total PCBs, total DDT, and methyl mercury for the green heron (Table 7-33). The largest HQ calculated for this area was for total DDT (HQ of 93).

Table 7-33. Green Heron Dose Compared to TRV Values for the Ship Channel

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww [a]	Dose (:g/gbw/d) [b]	TRV	HQ
Dioxins and furans						
2378-TCDD	na	na	na		1.4E-05	
2378-TCDF	na	na	na		1E-06	
PCBs						
Total PCB	4.7	1.43	0.5025	1.264	0.41	**3.08**
Pesticides						
Dieldrin	0.04	0.003	0.00075	0.011	0.077	0.14
Heptachlor Epoxide	0	0.002	0.0006	0.00004	na	
Lindane	0	0.001	0.0003	0.00002	2	0.00001
Total DDT	1.05	0.082	0.022	0.278	0.003	**92.62**
Trace Elements						
Arsenic	na				2.5	
Cadmium	na	0.662	0.497	0.014	1.45	0.01
Lead	na	726.000	544.5	14.941	1.13	**13.22**
Mercury	na	1.843	1.383	0.038	0.45	0.08
Methyl mercury	na	1.843	1.383	0.038	0.0006	**63.23**
Selenium	na		na		0.5	
Zinc	na	218.000	163.5	4.486	14.5	0.31

[a] calculated from dry weight concentrations assuming 85 percent moisture

[b] based on diet of 94 percent fish, 6 percent invertebrates, and 1.8 percent incidental sediment ingestion, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day

7.6 AQUATIC MAMMAL EVALUATION

7.6.1 SCREENING LEVEL EXPOSURE ESTIMATE

Several different dietary regimes were reported for the raccoon in U.S. EPA, 1993, the dietary regime with the greatest aquatic component was selected for the screening assessment. Therefore, the raccoon dietary composition used for this estimate corresponds to the diet observed for raccoons feeding in tidal mudflats in southwestern Washington (Tyson, 1950). In addition to the consumption of fish and invertebrates, the incidental ingestion of sediment associated with the consumption of invertebrates will also be considered (Table 7-34).

Table 7-34. Dietary Composition for Raccoon (U.S. EPA 1993)

SPECIES	PERCENT FISH	PERCENT INVERTEBRATES	PERCENT INCIDENTAL SEDIMENT INGESTION
Raccoon	**10**	**90**	**9.4**

The normalized ingestion rate for the raccoon was calculated using the approach described in Section 5. The following values were used for the raccoon (non-herbivorous mammal) calculations:

$$FMR_{(kcal/day)} = 0.6167(BW)^{0.862}$$

Eq. 7-6

Mean body weight is 5.8 kg for the raccoon. For this risk assessment, body weights were calculated by taking the average of the mean male and female body weights.

The estimated normalized ingestion rate for the raccoon was 0.17g/g body weight/day.

7.6.2 DIOXINS AND FURANS TEQ EVALUATION

TEFs have been developed which can be used to express the toxicity of individual dioxin and furan concentrations in terms of the equivalent toxicity of a particular concentration of 2,3,7,8-TCDD. A number of TEF values have been developed to calculate 2,3,7,8-TCDD TEQs for fish, birds and mammals. The calculated TEQ dose for the raccoon was evaluated using TRVs calculated from a study conducted by Heaton et al. (1995). In this study, adult mink were fed carp collected from the mouth of the Saginaw River and were then evaluated for reproductive effects. Even at the lowest dietary dose, significant reductions in gestation duration, kit body weight, and kit survival at 3 and 6 weeks of age were seen. The NOAEL was generated using the control group data.

The maximum fish tissue concentration reported for either the largemouth bass or the brown bullhead for each dioxin and furan congener was selected for use in this screening evaluation. The congener concentrations, TEF values and calculated TEQ values for the upper river and lower river zones are presented in Tables 7-35 and 7-36, respectively.

Table 7-35. WHO TEF Values for Mammals and Calculated TEQ Values Based on the Maximum Fish Tissue Concentrations for the Upper River

	Congeners	Mammals WHO TEF	Fish tissue max conc ng/kg	TEQ
Dioxins				
	2,3,7,8-TCDD	1	0.45	0.45
	1,2,3,78-PeCDD	1	0.55	0.55
	1,2,3,4,7,8-HxCDD	0.1	1.05	0.105
	1,2,3,6,7,8-HxCDD	0.1	na	
	1,2,3,7,8,9-HxCDD	0.1	na	
	1,2,3,4,6,7,8-HpCDD	0.01	0.8	0.008
	OCDD	0.0001	na	
Furans				
	2,3,7,8-TCDF	0.1	0.7	0.07
	1,2,3,7,8-PeCDF	0.05	5	0.25
	2,3,4,7,8-PeCDF	0.5	4.75	2.375

1,2,3,4,7,8-HxCDF	0.1	0.6	0.06
1,2,3,6,7,8-HxCDF	0.1	na	
1,2,3,7,8,9-HxCDF	0.1	na	
2,3,4,6,7,8-HxCDF	0.1	0.95	0.095
1,2,3,4,6,7,8-HpCDF	0.01	13.5	0.135
1,2,3,4,7,8,9-HpCDF	0.01	na	
OCDF	0.0001	31.5	0.00315
		SUM TEQ	4.10115

Table 7-36. WHO TEF Values for Mammals and Calculated TEQ Values Based on the Maximum Fish Tissue Concentrations for the Lower River

	Congeners	Mammals WHO TEF	Fish tissue max conc ng/kg	TEQ
Dioxins				
	2,3,7,8-TCDD	1	2.8	2.8
	1,2,3,78-PeCDD	1	3.9	3.9
	1,2,3,4,7,8-HxCDD	0.1	1.5	0.15
	1,2,3,6,7,8-HxCDD	0.1	4.9	0.49
	1,2,3,7,8,9-HxCDD	0.1	na	
	1,2,3,4,6,7,8-HpCDD	0.01	6.2	0.062
	OCDD	0.0001	57.1	0.00571
Furans				
	2,3,7,8-TCDF	0.1	2.4	0.24
	1,2,3,7,8-PeCDF	0.05	3	0.15
	2,3,4,7,8-PeCDF	0.5	4.6	2.3
	1,2,3,4,7,8-HxCDF	0.1	2.5	0.25
	1,2,3,6,7,8-HxCDF	0.1	1.6	0.16
	1,2,3,7,8,9-HxCDF	0.1	3.6	0.36
	2,3,4,6,7,8-HxCDF	0.1	0.2	0.02
	1,2,3,4,6,7,8-HpCDF	0.01	1.85	0.0185
	1,2,3,4,7,8,9-HpCDF	0.01	2.55	0.0255
	OCDF	0.0001	17.7	0.00177
			SUM TEQ	10.93348

The TEQ dose, TRV values, and calculated HQ values for raccoon in both the upper river and lower river zones are presented in Table 7-37. HQs greater than one were calculated for the raccoon in both the upper and lower river zones using the NOAEL TEQ value as the TRV. The largest HQ value (HQ: 12.2) was calculated for the raccoon in the lower river. When the LOAEL TEQ values were used as TRV values, all the resulting HQ values were less than one.

Table 7-37. Comparison of 2,3,7,8-TCDD TEQs to NOAEL Doses

		TEQ	Ingestion	Dose	NOAEL TEQ	HQ	LOAEL TEQ	HQ
Upper River								
	Raccoon	4.10	0.17	0.70	0.153	**4.56**	3.757	0.19
Lower River								
	Raccoon	10.93	0.17	1.86	0.153	**12.15**	3.757	0.49

7.6.3 SCREENING LEVEL EFFECTS ASSESSMENT

The estimated wildlife NOAELs for mink reported by Sample et al. (1996) were used as TRV values for the raccoon. The TRV values and the calculated dose for the raccoon are presented in Tables 7-38, 7-39, and 7-40 for the upper river zone, the lower river zone, and the Ship Channel/Tidal Basin zone. The speciation of mercury in the fish tissues was not reported. Therefore, the dose of mercury to each species was evaluated twice, first assuming that the concentration was entirely inorganic mercury, then assuming the concentration was entirely methyl mercury.

In the upper river zone, HQs greater than one were calculated for the estimated dose of total PCBs, arsenic, lead, and methyl mercury to the raccoon (Table 7-38). In the lower river zone, HQs greater than one were calculated for total PCBs, arsenic, cadmium, lead, mercury, methyl mercury, and zinc in the raccoon (Table 7-39). The largest HQs were calculated for arsenic, cadmium, and lead in raccoons with and HQ of 2,660 for lead in raccoons.

Table 7-38. Raccoon Dose Compared to TRV Values for Upper River

	Fish Tissue Max conc. (ug/g ww)	Invertebrates ug/g ww	Sediment ug/g ww [a]	Dose (ug/gbw/d) [b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.0000005	na	na	0.00000001	0.0000008	0.01
2378-TCDF	0.0000007	na	na	0.00000001	na	
PCBs						
Total PCB [c]	0.97	0.751	0.2445	0.135	0.069	**1.96**
Pesticides						
Dieldrin	0.0095	0.003	0.00075	0.001	0.015	0.04
Heptachlor Epoxide	0.0001	0.002	0.0006	0.0003	0.1	0.0032
Lindane	0.00031	0.001	0.0003	0.000	6.15	0.00003
Total DDT	0.0901	0.082	0.022	0.014	0.62	0.02
Trace Elements						
Arsenic	0.034	1.292	0.969	0.214	0.052	**4.11**
Cadmium	0.0057	0.524	0.393	0.087	0.742	0.1166

Lead	0.104	44.8	33.6	7.393	6.15	**1.2021**
Mercury	0.051	0.118	0.0885	0.020	1	0.02
Methyl mercury	0.051	0.118	0.0885	0.020	0.015	**1.36**
Selenium	0.193	nd	nd	0.003	0.154	0.02
Zinc	5.92	89.4	71.55	14.922	123.1	0.12

[a] calculated from dry weight concentrations assuming 85 percent moisture
[b] based on diet of 10 percent fish, 90 percent invertebrates and 9.4 percent incidental sediment ingestion (EPA 1993), body weight of 5.8 Kg and a calculated ingestion rate of 0.17g/g body weight/day
[c]Total PCB TRV based on Aroclor 1242 in mink

Table 7-39. Raccoon Dose Compared to TRV Values for Lower River

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww [a]	Dose (ug/gbw/d) [b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.00001	na	na	0.00000017	0.0000008	0.2125
2378-TCDF	0.0000024	na	na	0.00000004	na	
PCBs						
Total PCB [c]	1.27	5.805	1.8	0.939	0.069	**13.60**
Pesticides						
Dieldrin	0.02	0.002	0.000675	0.001	0.015	0.04
Heptachlor Epoxide	0.017	0.00035	0.00009	0.0003	0.1	0.0034
Lindane	0.0012	0.001	0.000135	0.0002	6.15	0.00003
Total DDT	0.28	0.004	0.001	0.0054	0.62	0.01
Trace Elements						
Arsenic	0.099	5.38	4.04	0.89	0.052	**17.10**
Cadmium	0.0041	0.636	0.477	0.11	0.742	0.14
Lead	0.043	115	116	19.45	6.15	**3.16**
Mercury	0.078	0.54	0.405	0.09	1	0.09
Methyl mercury	0.078	0.54	0.405	0.09	0.015	**6.03**
Selenium	0.27	0.22	0.165	0.04	0.154	0.27
Zinc	4.56	102.4	76.8	16.97	123.1	0.14

[a] calculated from dry weight concentrations assuming 85 percent moisture
[b] based on diet of 10 percent fish, 90 percent invertebrates and 9.4 percent incidental sediment ingestion (EPA 1993), body weight of 5.8 Kg and a calculated ingestion rate of 0.17g/g body weight/day
[c]Total PCB TRV based on Aroclor 1242 in mink

Finally, in the Washington Ship Channel/Tidal Basin zone, HQs greater than one were calculated for total PCBs, arsenic, lead, and methyl mercury in the raccoon (Table 7-40). The largest HQ value was calculated for arsenic and PCBs with HQs of 4.1 and 4.5, respectively.

Table 7-40. Raccoon Dose Compared to TRV Values for Ship Channel

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww [a]	Dose (:g/gbw/d) [b]	TRV	HQ
Dioxins and furans						
2378-TCDD	na	na	na	na	0.0000008	
2378-TCDF	na	na	na	na	na	
PCBs						
Total PCB [c]	4.7	1.43	0.5025	0.307	0.069	**4.45**
Pesticides						
Dieldrin	0.04	0.003	0.00075	0.001	0.015	0.08
Heptachlor Epoxide	0	0.002	0.0006	0.0003	0.1	0.003
Lindane	0	0.001	0.0003	0.0002	6.15	0.00003
Total DDT	1.05	0.082	0.022	0.031	0.62	0.05
Trace Elements						
Arsenic	na					
Cadmium	na	0.662	0.497	0.109	0.742	0.15
Lead	na	726	544.5	119.779	6.15	**19.48**
Mercury	na	1.843	1.383	0.304	1	0.30
Methyl mercury	na	1.843	1.383	0.304	0.015	**20.27**
Selenium	na	na	na		0.154	
Zinc	na	218	163.5	35.967	123.1	0.29

[a] calculated from dry weight concentrations assuming 85 percent moisture
[b] based on diet of 10 percent fish, 90 percent invertebrates and 9.4 percent incidental sediment ingestion (EPA 1993), body weight of 5.8 Kg and a calculated ingestion rate of 0.17g/g body weight/day
[c]Total PCB TRV based on Aroclor 1242 in mink

7.7 UNCERTAINTY ANALYSIS

The lack of sediment dioxin and furan concentrations resulted in uncertainty in the benthic invertebrate, plus the aquatic bird and mammal evaluations. Sediment dioxin and furan data was not available to be screened against criteria for the benthic invertebrates. In addition, benthic prey tissue concentrations of dioxins and furans could not be estimated in the aquatic bird and mammal evaluation due to the lack of sediment data.

Amphibians were not evaluated as potential receptors. Amphibians are present within the Anacostia River. Recent research has shown that effects in frogs can occur at concentrations below AWQC for nitrite (Marco et al, 1999). Further research is required to develop exposure parameters and criteria appropriate for this receptor group before screening level assessments can be conducted. Without this information it is difficult to assess the potential for risk to amphibians in the context of a screening level assessment.

7.7.1 BENTHIC INVERTEBRATES EVALUATION

Sediment toxicity data was taken from only two studies, both of which used the same test organism, the amphipod *Hyalella azteca*. Therefore, there is some uncertainty associated with this evaluation and it cannot be considered a definitive assessment of sediment toxicity within the lower Anacostia. The sparse distribution of sampling locations for these bioassay studies was also not sufficient to adequately characterize the entire study area or to delineate regions within the study area as toxic or non-toxic.

The comparison of maximum sediment concentrations to sediment criteria was conducted in order to assess risk for benthic invertebrates. The use of maximum concentrations provides a very conservative estimate of exposure in the Anacostia River sediments. In addition, there may be some uncertainty associated with the use of sediment benchmarks that do not explicitly address many of the factors that can affect the bioavailability of the sediment contaminants, such as, the speciation of trace elements, the presence or absence of acid volatile sulfides (AVS), or the organic carbon content of the sediments which can affect the availablility of organic contaminants. For such uncertainty to have a major influence on the conclusions of this screening level estimate, bioavailability of contaminants would have to be shown to be substantially less than that predicted. Since bioavailability of COPCs is corroborated by tissue residue measurements, this assumption does not appear unreasonable. Therefore, these uncertainty factors have marginal bearing on the results of this screening.

7.7.2 FISH EVALUATION

Fish species were evaluated in three ways: the surface water contaminant concentrations were compared to AWQC values; fish tissue concentrations were compared to tissue residue effects concentrations; and sediment PAH concentrations were screened against sediment criteria associated with the occurance of neoplams and reproductive impairment in fish. The uncertainties associated with each of these approaches is discussed.

The surface water data that was compared to AWQC values represented only one study using discrete samples collected seven times throughout 1998. This data does not provide a basis for determining the exposure of fish over a longer period of time. The criteria values are generally developed to be protective of an average concentration over a set time period (e.g., 4 hours or 14 days).

Using tissue residue effects concentrations in fish to evaluate the potential for risk available in the published literature is complicated by a number of factors including:

X Differences in species sensitivity

X Differences in the tissues analyzed

X Differences in exposure scenarios in laboratory and field studies

X Lack of consistency in the endpoints evaluated

X Differences in the species of the contaminant (e.g., inorganic mercury vs. methylmercury)

Finally, there is some uncertainty associated with the exposure assessment used to screen sediment PAH concentrations. Although PAHs are known to cause adverse effects in fish, methodologies to quantify

this risk are still being developed. Some researchers have used PAH concentrations in sediment associated with elevated rates of neoplasia and reproductive impairment in fish as an evaluation value, but this approach is subject to confounding factors such as the presence of co-occurring contaminants in the field. In addition, the database that was used for this purpose contained a comparatively small number of data points. The ecological significance of neoplasms in fish populations has yet to be firmly established, and no further information is currently published for determining sediment effects concentrations for other, possibly more sensitive endpoints such as reproductive impairment and immune dysfunction. Baumann et al. (1990) reported that in the Black River, a system with a high prevalence of liver tumors, there were few fish of age 5 and no fish of age 6 or 7. In an uncontaminated reference system age 6 and 7 fish comprised 18 percent of the sampled population. The authors hypothesized that there was an age-selective mortality associated with a high prevalence of liver tumors.

Finally, the movement of fish populations introduces another source of uncertainty in the fish evaluation. Fish collected in the Anacostia River could have been exposed elsewhere. Therefore, tissue concentrations and tumors present in these fish could reflect exposure conditions outside the Anacostia.

7.7.3 AQUATIC BIRD AND MAMMAL EVALUATION

The use of generic equations to estimate the normalized ingestion rate, the field metabolic rate, and the estimated average metabolizable energy of bird and mammal diets provide reasonable estimates of these values for the calculation of normalized ingestion rates for the receptor species in the absence of species-specific data. However, there is some uncertainty with regard to the resulting estimated species-specific ingestion rates.

In addition, the use of BSAF values to estimate invertebrate tissue concentrations introduces some uncertainty into the estimated dose calculation. The BSAF values that were used were average values derived from an extremely large data set. The use of an average value for entire classes of compounds, such as PCBs and pesticides, simplifies the complex chemistry of the individual compounds within the contaminant class. For the purposes of this assessment, the BSAFs provide a reasonable estimate of concentrations in benthic invertebrates resulting from exposure to sediment contaminants. However, site-specific conditions could influence the uptake of contaminants by invertebrates in the Anacostia River.

Uncertainties due to interspecific differences in toxicity of the COPCs were not addressed in the development of the TRV values or in the risk calculation. Toxicity data for ducks, chicken, kestrel, pheasant, barn owl, and pelicans were used to derive TRV values for great blue heron (Sample et al., 1996). These TRV values were then used to calculate risks to the green heron. Similarly, for the mammal assessment, toxicity data for rats, mice, and mink were used to derive TRV values for mink (Sample et al., 1996). These TRV values were then used to calculate risks for the raccoon.

7.8 ADDITIONAL EVALUATIONS

Screening level risk assessments are simplified assessments that can be conducted with limited data, using conservative assumptions for the parameters for which site-specific data are lacking (U.S. EPA, 1997). Conservative approaches are used to minimize the potential of failing to identify a potential risk. This approach ensures that sites that have the potential to pose risks to ecological receptors are further evaluated in a baseline risk assessment. Such a conservative approach may use unrealistic assumptions though. For instance, it is unrealistic to assume that 100% of ingested contaminants are assimilated upon ingestion. Before a complete baseline risk assessment is conducted, reassessment using more realistic parameters should be conducted to help focus future efforts.

Additional evaluations were conducted for benthic invertebrates, fish, birds, and mammals using more realistic assumptions than those used in the evaluations described in Sections 7.3, 7.4, 7.5, and 7.6. For

example, evaluations were conducted using mean sediment contaminant concentrations in addition to the maximum concentrations. The results of these evaluations are presented in the following sections.

When dose estimates are derived from average exposure point estimates for the Upper and Lower river using the alternate diet (Tables 7-40a and 7-40b, respectively) only arsenic exceeds the TRV benchmark (HQs of 1.35 and 2.12, respectively).

Table 7-40a. Alternate Raccoon Dose Based on Average Exposure Compared to TRV Values for Upper River

	Fish Tissue average conc. (ug/g ww)	Invertebrates ug/g ww	Sediment ug/g ww[a]	Dose (ug/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.000000450	na	na	0.000000002	0.0000008	0.003
2378-TCDF	0.000000625	na	na	0.000000003	na	
PCBs						
Total PCB [c]	0.531	0.303123596	0.0987	0.023	0.069	0.34
Pesticides						
Dieldrin	0.008	0.001	0.0003	0.00011	0.015	0.01
Heptachlor Epoxide	0.002	0.001	0.0003	0.00008	0.1	0.0008
Lindane	0.00021	0.0006	0.00015	0.00004	6.15	0.00001
Total DDT	0.08	0.044	0.012	0.003	0.62	0.01
Trace Elements						
Arsenic	0.034	0.938	0.7035	0.070	0.052	**1.35**
Cadmium	0.0047	0.35	0.263	0.026	0.742	0.035
Lead	0.019	31.2	23.4	2.337	6.15	0.380
Mercury	0.037	0.058	0.0435	0.005	1	0.005
methyl mercury	0.037	0.058	0.0435	0.005	0.015	0.30
Selenium	0.14	nd	nd	0.001	0.154	0.00
Zinc	3.93	71	53.3	5.338	123.1	0.04

[a] calculated from dry weight concentrations assuming 85 percent moisture
[b] based on diet of 3 percent fish, 37 percent invertebrates and 9.4 percent incidental sediment ingestion, body weight of 5.8 Kg and a calculated ingestion rate of 0.17 g/g body weight/day (EPA 1993)
[c] Total PCB TRV based on Aroclor 1242 in mink

Table 7-40b. Alternate Raccoon Dose Based on Average Exposure Compared to TRV Values for Lower River

	Fish Tissue Ave. conc. (ug/g ww)	Invertebrates ug/g ww	Sediment ug/g ww[a]	Dose (ug/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.00000245	na	na	0.000000012	0.0000008	0.02
2378-TCDF	0.0000014	na	na	0.000000007	na	
PCBs						
Total PCB [c]	0.703	0.595044248	0.184	0.044	0.069	0.64
Pesticides						
Dieldrin	0.01000	0.000578171	0.00015	0.00009	0.015	0.01
Heptachlor Epoxide	0.00580	0.000173451	0.000045	0.00004	0.1	0.00041
Lindane	0.000513	0.000	0.00006	0.00002	6.15	0.000003
Total DDT	0.174	0.058	0.015	0.005	0.62	0.01
Trace Elements						
Arsenic	0.09902	1.462	1.1	0.110	0.052	**2.12**
Cadmium	0.00413	0.292	0.219	0.022	0.742	0.03
Lead	0.04332	38.8	29.1	2.906	6.15	0.47
Mercury	0.078	0.112	0.084	0.009	1	0.01
Methyl mercury	0.078	0.112	0.084	0.009	0.015	0.59
Selenium	0.27	0.146	0.11	0.012	0.154	0.08
Zinc	4.56	54.8	41.1	4.127	123.1	0.03

[a] calculated from dry weight concentrations assuming 85 percent moisture
[b] based on diet of 3 percent fish, 37 percent invertebrates and 9.4 percent incidental sediment ingestion, body weight of 5.8 Kg and a calculated ingestion rate of 0.17 g/g body weight/day (EPA 1993)
[c]Total PCB TRV based on Aroclor 1242 in mink

7.8.1 BENTHIC INVERTEBRATES

In addition to screening the maximum sediment contaminant concentrations, the mean sediment concentrations were screened (Table 7-41). The screening was conducted using two sediment benchmark values, TELs and Probable Effects Levels (PELs). PELs are differentiated from TEL values in that they are intended to define threshold values above which toxicity is highly probable.

PELs were derived by calculating the geometric mean of the 50th percentile of the effect data set and the 85th percentile of the no-effect dataset. PELs were intended to estimate the sediment concentration of a chemical above which adverse biological effects frequently occurred (Smith et al., 1996). Most of the PELs presented in Table 7-41 were obtained from Smith et al. (1996). The PELs reported for LPAH, HPAH, and total PAH concentrations were taken from the ARCS data for the 28-day exposure of *Hyalella azteca* to freshwater sediments (U.S. EPA, 1996).

Table 7-41. Sediment Benchmarks

	TEL	PEL
Trace elements		
Arsenic	10.8	17

	Barium [(a)]	0.7	na
	Cadmium	0.6	3.53
	Chromium, total	36.3	90
	Copper	28	197
	Lead	34.2	91.3
	Manganese	615	na
	Mercury	0.17	0.486
	Nickel	19.5	35.9
	Selenium [(a)]	0.29	na
	Silver [(a)]	<0.5	na
	Strontium [(a)]	49	na
	Vanadium [(a)]	50	na
	Zinc	94.2	315
PAHs			
	Benzo(a) anthracene	0.032	0.385
	Benzo(a)pyrene	0.032	0.782
	Chrysene	0.032	0.862
	Fluoranthene	0.11	2.35
	Phenanthrene	0.042	0.515
	Pyrene	0.053	0.875
	LPAHs	0.076	1.17
	HPAHs	0.193	2.34
	Total PAHs	0.264	3.37
PCBs			
	Aroclor 1254	0.032	0.277
	Aroclor 1260	0.032	0.277
	Total PCBs	0.032	0.277
Pesticides			
	Chlordane	0.0045	0.0089
	Dieldrin	0.0029	0.0067
	Heptaclor Epoxide	0.0006	0.00274
	Lindane	0.0009	0.00138
	DDD	0.00354	0.00851
	DDE	0.0014	0.00675
	total DDT	0.007	4.45
	Endrin	0.00267	0.0624

[(a)] background freshwater sediment values (Buchman 1999)

The HQ values calculated from the screening of maximum and mean sediment concentrations for the upper river zone are presented in Table 7-42. TEL-HQs greater than one were calculated for both maximum and mean concentrations of most of the trace elements, PAHs, PCBs and pesticides. The largest TEL-HQ values were calculated for barium and total PCB concentrations. Sediment concentrations were compared to the corresponding PEL values to calculate PEL-HQ values. PEL-HQs greater than one were calculated for chromium, lead, mercury, nickel, and zinc, as well as most of the PAH compounds, total PCBs, and many of the organochlorine pesticides. The largest PEL-HQ was calculated for the maximum chlordane concentration (HQ of 22).

Table 7-42. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Upper River

	TEL	PEL	Sediment concentration		n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
Trace elements							
Arsenic	10.8	17	max	6.46		0.60	0.38
			mean	4.69	8	0.43	0.28
Barium [(a)]	0.7	na	max	156.40		**223.43**	
						172.14	
Cadmium	0.6	3.53	max	2.62		**4.37**	0.74
			mean	1.75	20	**2.91**	0.50
Chromium, total	36.3	90	max	134.00		**3.69**	**1.49**
			mean	82.46	13	**2.27**	0.92
Copper	28	197	max	100.10		**3.58**	0.51
			mean	60.63	13	**2.17**	0.31
Lead	34.2	91.3	max	224.00		**6.55**	**2.45**
			mean	156.00	20	**4.56**	**1.71**
Manganese	615	na	max	643.10		**1.05**	
			mean	471.49	8	0.77	
Mercury	0.17	0.486	max	0.59		**3.47**	**1.21**
			mean	0.29	20	**1.72**	0.60
Nickel	19.5	35.9	max	50.51		**2.59**	**1.41**
			mean	41.96	8	**2.15**	**1.17**
Selenium [(a)]	0.29	na	max	nd			
			mean				
Silver [(a)]	< 0.5	na	max	nd			
			mean				
Strontium [(a)]	49	na	max	21.72		0.44	
			mean	16.48	8	0.34	
Vanadium [(a)]	50	na	max	65.54		**1.31**	
			mean	51.31	8	**1.03**	
Zinc	94.2	315	max	477.00		**5.06**	**1.51**
			mean	355.27	20	**3.77**	**1.13**
PAHs							
Benzo(a) anthracene	0.032	0.385	max	0.78		**24.41**	**2.03**
			mean	0.56	13	**17.47**	**1.45**
Benzo(a)pyrene	0.032	0.782	max	0.70		**21.72**	0.89
			mean	0.51	13	**16.03**	0.66
Chrysene	0.032	0.862	max	1.10		**34.38**	**1.28**
			mean	0.802	13	**25.06**	0.93
Fluoranthene	0.11	2.35	max	1.79		**16.27**	0.76
			mean	0.936	13	**8.51**	0.40
Phenanthrene	0.042	0.515	max	0.85		**20.24**	**1.65**
			mean	0.557	13	**13.26**	**1.08**
Pyrene	0.053	0.875	max	1.58		**29.81**	**1.81**
			mean	0.840	13	**15.85**	0.96
LPAHs	0.076	1.17	max	1.68		**22.11**	**1.44**
			mean	1.075	13	**14.14**	0.92
HPAHs	0.193	2.34	max	6.60		**34.20**	**2.82**

Table 7-42. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Upper River

		TEL	PEL	Sediment concentration		n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
				mean	4.450	13	**23.06**	**1.90**
	Total PAHs	0.264	3.37	max	7.89		**29.89**	**2.34**
				mean	5.520	13	**20.91**	**1.64**
PCBs								
	Aroclor 1254	0.032	0.277	max	1.63		**50.94**	**5.88**
				mean	0.671	8	**20.97**	**2.42**
	Aroclor 1260	0.032	0.277	max	0.02		0.47	0.05
				mean	0.008	7	0.26	0.03
	Total PCBs	0.032	0.277	max	1.63		**50.94**	**5.88**
				mean	0.658	19	**20.56**	**2.38**
Pesticides								
	Chlordane	0.0045	0.0089	max	0.20		**43.56**	**22.02**
				mean	0.146	13	**32.44**	**16.40**
	Dieldrin	0.0029	0.0067	max	0.01		**1.72**	0.75
				mean	0.002	16	0.59	0.25
	Heptaclor Epoxide	0.0006	0.00274	max	0.004		**6.67**	**1.46**
				mean	0.002	3	**2.67**	0.58
	Lindane	0.0009	0.00138	max	0.002		**2.22**	**1.45**
				mean	0.001	5	0.62	0.40
	DDD	0.00354	0.00851	max	0.08		**23.16**	**9.64**
				mean	0.037	19	**10.45**	**4.35**
	DDE	0.0014	0.00675	max	0.05		**33.57**	**6.96**
				mean	0.027	19	**19.29**	**4.00**
	total DDT	0.007	4.45	max	0.15		**21.29**	0.03
				mean	0.080	27	**11.43**	0.02
	Endrin	0.00267	0.0624	max	0.00		**1.12**	0.05
				mean	0.001	2	0.37	0.02

[(a)] background freshwater sediment values (Buchman 1999)

In the lower river zone, the largest TEL-HQs were reported for PAH concentrations (Table 7-43). TEL-HQs greater than one were reported for both the maximum and mean concentrations of most of the trace elements, all of the PAHs, PCBs, and many of the pesticides. The largest PEL-HQs were also reported for the PAH compounds. All of the PEL-HQs calculated for maximum and mean PAH concentrations were greater than one and PEL-HQs greater than one hundred were reported for the maximum concentrations of benzo(a)anthracene, phenanthrene, and pyrene.

Table 7-43. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Lower River

	TEL	PEL	Sediment concentration	n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
Trace elements						

Table 7-43. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Lower River

	TEL	PEL		Sediment concentration	n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
Arsenic	10.8	17	max	26.90		**2.49**	**1.58**
			mean	7.31	22	0.68	0.43
Barium [(a)]	0.7	na	max	170.00		**242.86**	
			mean	111.73	22	**159.62**	
Cadmium	0.6	3.53	max	3.18		**5.30**	0.90
			mean	1.46	30	**2.43**	0.41
Chromium, total	36.3	90	max	155.50		**4.28**	**1.73**
			mean	62.32	37	**1.72**	0.69
Copper	28	197	max	631.00		**22.54**	**3.20**
			mean	102.62	37	**3.67**	0.52
Lead	34.2	91.3	max	775.00		**22.66**	**8.49**
			mean	193.98	37	**5.67**	**2.12**
Manganese	615	na	max	800.00		**1.30**	
			mean	448.87	23	0.73	
Mercury	0.17	0.486	max	2.70		**15.88**	**5.56**
			mean	0.56	29	**3.27**	**1.14**
Nickel	19.5	35.9	max	69.70		**3.57**	**1.94**
			mean	33.24	23	**1.70**	0.93
Selenium [(a)]	0.29	na	max	1.10		**3.79**	
			mean	0.73	2	**2.50**	
Silver [(a)]	<0.5	na	max	64.40		**128.80**	
			mean	6.57	14	**13.14**	
Vanadium [(a)]	50	na	max	68.10		**1.36**	
			mean	34.34	22	0.69	
Zinc	94.2	315	max	512.00		**5.44**	**1.63**
			mean	273.77	37	**2.91**	0.87
PAHs							
Benzo(a) anthracene	0.032	0.385	max	100.00		**3125.00**	**259.74**
			mean	5.08	46	**158.69**	**13.19**
Benzo(a)pyrene	0.032	0.782	max	27.00		**843.75**	**34.53**
			mean	2.89	44	**90.34**	**3.70**
Chrysene	0.057	0.862	max	86.00		**1508.77**	**99.77**
			mean	5.03	48	**88.30**	**5.84**
Fluoranthene	0.11	2.35	max	110.00		**1000.00**	**46.81**
			mean	7.86	49	**71.45**	**3.34**
Phenanthrene	0.042	0.515	max	360.00		**8571.43**	**699.03**
			mean	17.23	48	**410.24**	**33.46**
Pyrene	0.053	0.875	max	320.00		**6037.74**	**365.71**
			mean	13.96	49	**263.40**	**15.95**
LPAHs	0.076	1.17	max	98.80		**1300.00**	**84.44**
			mean	13.10	33	**172.37**	**11.20**
HPAHs	0.193	2.34	max	127.00		**658.03**	**54.27**
			mean	19.10	37	**98.96**	**8.16**
Total PAHs	0.264	3.37	max	211.00		**799.24**	**62.61**

Table 7-43. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Lower River

	TEL	PEL		Sediment concentration	n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
			mean	30.78	37	**116.59**	**9.13**
PCBs							
Aroclor 1254	0.032	0.277	max	nd			
			mean				
Aroclor 1260	0.032	0.277	max	12.00		**375.00**	**43.32**
			mean	2.13	6	**66.50**	**7.68**
Total PCBs	0.032	0.277	max	12.00		**375.00**	**43.32**
			mean	1.23	17	**38.44**	**4.44**
Pesticides							
Chlordane	0.0045	0.0089	max	0.1400		**31.11**	**15.73**
			mean	0.0650	15	**14.44**	**7.30**
Dieldrin	0.0029	0.0067	max	0.0050		**1.72**	0.75
			mean	0.0010	14	0.34	0.15
Heptaclor Epoxide	0.0006	0.00274	max	0.0003		0.53	0.12
			mean	0.0003	1	0.53	0.12
Lindane	0.0009	0.00138	max	0.0010		**1.11**	0.72
			mean	0.0004	5	0.43	0.28
DDD	0.00354	0.00851	max	0.1710		**48.31**	**20.09**
			mean	0.0074	14	**2.09**	0.87
DDE	0.0014	0.00675	max	0.0730		**52.14**	**10.81**
			mean	0.0005	8	0.38	0.08
total DDT	0.007	4.45	max	0.3240		**46.29**	0.07
			mean	0.0957	14	**13.67**	0.02
Endrin	0.00267	0.0624	max	0.0034		**1.28**	0.05
			mean	0.0013	8	0.49	0.02

[(a)] background freshwater sediment values (Buchman 1999)

In the Ship Channel zone, TEL-HQs greater than one were calculated for both the maximum and mean concentrations of all the trace elements, the PAHs, the PCBs, and most of the pesticides (Table 7-44). The highest TEL-HQ values were reported for the PAH compounds. PEL-HQ values greater than one were calculated for most of the trace elements, PAHs, PCBs and several pesticides (chlordane, dieldrin, heptaclor epoxide, lindane, DDD, and DDE).

Table 7-44. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Ship Channel

	TEL	PEL		Sediment concentration	n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
Trace elements							
Arsenic	10.8	17	max	nd			

Table 7-44. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Ship Channel

	TEL	PEL	Sediment concentration		n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
			mean				
Barium [a]	0.7	na	max	nd			
			mean				
Cadmium	0.6	3.53	max	3.31		5.52	0.94
			mean	1.12	20	1.86	0.32
			mean				
Chromium, total	36.3	90	max	176.00		4.85	1.96
			mean	94.06	20	2.59	1.05
			mean				
Copper	28	197	max	348.00		12.43	1.77
			mean	89.64	20	3.20	0.46
			mean				
Lead	34.2	91.3	max	3630.00		106.14	39.76
			mean	498.54	20	14.58	5.46
			mean				
Manganese	615	na	max	nd			
			mean				
Mercury	0.17	0.486	max	9.22		54.21	18.96
			mean	0.79	20	4.67	1.63
			mean				
Nickel	19.5	35.9	max	nd			
			mean				
Selenium [a]	0.29	na	max	nd			
			mean				
Silver [a]	<0.5	na	max	nd			
			mean				
Vanadium [a]	50	na	max	nd			
			mean				
Zinc	94.2	315	max	1090.00		11.57	3.46
			mean	340.78	20	3.62	1.08
			mean				
PAHs							
Benzo(a) anthracene	0.032	0.385	max	8.98		280.63	23.32
			mean	0.9260	21	28.94	2.41

Table 7-44. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Ship Channel

	TEL	PEL	Sediment concentration		n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
Benzo(a)pyrene	0.032	0.782	max	6.48		202.50	8.29
			mean	0.7960	21	24.88	1.02
Chrysene	0.057	0.862	max	8.84		155.09	10.26
			mean	1.1610	21	20.37	1.35
Fluoranthene	0.11	2.35	max	19.71		179.18	8.39
			mean	2.2610	21	20.55	0.96
Phenanthrene	0.042	0.515	max	16.64		396.19	32.31
			mean	1.5930	21	37.93	3.09
Pyrene	0.053	0.875	max	14.61		275.66	16.70
			mean	1.8560	21	35.02	2.12
LPAHs	0.076	1.17	max	23.44		308.42	20.03
			mean	2.7640	21	36.37	2.36
HPAHs	0.193	2.34	max	65.98		341.87	28.20
			mean	8.1440	21	42.20	3.48
Total PAHs	0.264	3.37	max	89.41		338.67	26.53
			mean	10.9100	21	41.33	3.24
PCBs							
Aroclor 1254	0.032	0.277	max	nd			
			mean				
Aroclor 1260	0.032	0.277	max	nd			
			mean				
Total PCBs	0.032	0.277	max	3.35		104.69	12.09
			mean	0.8050		25.16	2.91
Pesticides							
Chlordane	0.0045	0.0089	max	0.13		28.89	14.61
			mean	0.0250	21	5.56	2.81
Dieldrin	0.0029	0.0067	max	0.01		3.21	1.39
			mean	0.0024	19	0.83	0.36
Heptaclor Epoxide	0.0006	0.00274	max	0.0028		4.67	1.02

Table 7-44. Sediment Benchmarks, Maximum Concentrations and Calculated Hazard Quotients for the Ship Channel

	TEL	PEL	Sediment concentration		n	$HQ_{(TEL)}$	$HQ_{(PEL)}$
			mean	0.0009	5	1.45	0.32
Lindane	0.0009	0.00138	max	0.0018		2.00	1.30
			mean	0.0004	16	0.44	0.29
DDD	0.00354	0.00851	max	0.20		55.65	23.15
			mean	0.0390	21	11.02	4.58
DDE	0.0014	0.00675	max	0.14		101.43	21.04
			mean	0.0430	21	30.71	6.37
total DDT	0.007	4.45	max	0.80		114.71	0.18
			mean	0.1290	21	18.43	0.03
Endrin	0.00267	0.0624	max	0.0015		0.56	0.02
			mean	0.0005	9	0.19	0.01

[(a)] background freshwater sediment values (Buchman 1999)

7.8.2 FISH

The fish receptors were evaluated in three ways: surface water concentrations were compared to AWQC, tissue concentrations were compared to tissue residue effects concentrations, and sediment PAH concentrations were compared to sediment benchmark concentrations. The additional evaluations consist of a comparison of mean surface water concentrations to AWQC values and a comparison of mean sediment PAH concentrations to sediment benchmark concentrations. Additional evaluations of the tissue concentrations were not conducted because many of the tissue residue effects concentrations were derived from LOEC values at which effects were seen in fish. It would not be appropriate to compare mean tissue concentrations to concentrations associated with effects as a screening evaluation.

The average surface water contaminant concentrations are presented in Table 7-45. The contaminants were selected based on the HQ values calculated in Section 7.5. Only those contaminants whose maximum concentrations exceeded the corresponding AWQC values were selected. In both the upper river zone and the lower river zone, mean ammonia concentrations exceeded the corresponding AWQC value (HQs of 1.7 in both areas). The mean concentrations of lead and total PCBs in the upper river zone did not exceed the corresponding AWQC.

Table 7-45. Average Surface Water Concentrations Compared to AWQC for Lower River Area and Upper River Area

		Mean Aqueous Conc.	n	AWQC	$HQ_{(AWQC)}$
Upper River					
	NH4-N	178.29	14	105.00	1.70
	Pb	0.85	14	2.50	0.34
	total PCBs	0.01	9	0.017	0.43
Lower River					
	NH4-N	179	43	105.00	1.70

The exposure of fish in the Anacostia River to PAHs was evaluated by comparing the maximum measured sediment PAH concentration to sediment benchmark concentrations.

In Table 7-46, the average PAH concentrations in each area is compared to the sediment benchmark of 2:g/g total PAH concentration. HQ values greater than one were calculated for all areas. The largest HQ value was calculated for the lower river zone (HQ of 15).

Table 7-46. Mean PAH Sediment Concentrations Compared to Sediment Benchmark Concentrations (:g/g)

LOCATION	MEAN PAH CONCENTRATION	SEDIMENT BENCHMARK	HQ
Upper river zone	5.52	2	2.8
Lower river zone	30.8	2	15
Ship channel	10.9	2	5.5

7.8.3 AQUATIC BIRDS AND MAMMALS

Additional evaluations of the green heron and raccoon were conducted. First, the average tissue and sediment concentrations were used to estimate doses to each of these species using the conservative dietary compositions presented in Sections 7.5 and 7.6. Then, an alternative exposure was calculated for the raccoon based on a less conservative dietary composition.

The average fish tissue concentrations were calculated using the fish tissue concentrations reported for the largemouth bass and the brown bullhead (Table 7-47). In addition, average sediment concentrations were used to calculate invertebrate tissue concentrations using the BSAF method presented in Section 7.6. These values were used to estimate dose to the green heron and raccoon using the dietary compositions presented in Section 7.6. The results of these calculations are compared to TRV values in Tables 7-48 through 7-53.

Table 7-47. Average Fish Tissue Concentrations (:g/g wet weight)

	Upper River	Lower River	Ship Channel
Dioxins and Furans:	n	n	n

2,3,7,8-TCDD	[a]	0.00000045	2	0.00000245	2		
2,3,7,8-TCDF		0.000000625	2	0.0000014	2		
PCBs:							
Total PCB		0.531	2	0.703	6	4.7	1
Trace Elements:							
Arsenic		0.0339	1	0.09902	1		
Cadmium	[a]	0.0047	3	0.00413	1		
Lead	[a]	0.0193	3	0.04332	1		
Mercury		0.0366	3	0.07839	1		
Selenium		0.1406	3	0.27438	1		
Zinc		3.9255	3	4.55923	1		
Pesticides:							
Dieldrin		0.008	6	0.01000	6	0.04000	1
Heptaclor Epoxide		0.002	5	0.00580	4		
Lindane		0.00021	3	0.000513	3		
DDD	[a]	0.037	6	0.077	6	0.36000	1
DDE	[a]	0.041	6	0.093	6	0.63000	1
DDT		0.08	6	0.174	6	0.06000	1

[a] Original LOEC was based on mortality endpoint, screening value = LOEC/10

In the upper river zone, HQ values greater than one were calculated for arsenic in the raccoon and, total DDT, and methyl mercury in the green heron using the average tissue and sediment concentrations (Table 7-48). The largest HQ value was calculated for methylmercury in the green heron (HQ of 18.2). Only one HQ greater than one was calculated for the raccoon (Table 7-49) for arsenic (HQ = 2.99)

Table 7-48. Green Heron Dose Based on Average Exposure Compared to TRV Values for the Upper River

	Fish Tissue average conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww [a]	Dose (:g/gbw/d) [b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.000000450	na	na	0.0000001	0.000014	0.01
2378-TCDF	0.000000625	na	na	0.0000002	0.000001	0.16
PCBs						
Total PCB	0.531	0.3031	0.0987	0.145	0.41	0.35
Pesticides						
Dieldrin	0.008	0.001	0.0003	0.002	0.077	0.03
Heptachlor Epoxide	0.002	0.001	0.0003	0.001	na	

Lindane	0.00021	0.0006	0.00015	0.0001	2	0.00003
Total DDT	0.08	0.044	0.012	0.022	0.003	**7.29**
Trace Elements						
Arsenic	0.034	0.938	0.7035	0.028	2.5	0.01
Cadmium	0.0047	0.35	0.263	0.008	1.45	0.01
Lead	0.019	31.2	23.4	0.647	1.13	0.57
Mercury	0.037	0.058	0.0435	0.011	0.45	0.02
Methyl mercury	0.037	0.058	0.0435	0.011	0.0006	**18.22**
Selenium	0.14	nd	nd	0.037	0.5	0.07
Zinc	3.93	71	53.3	2.496	14.5	0.17

(a) calculated from dry weight concentrations assuming 85 percent moisture
(b) based on diet of 94 percent fish, 6 percent invertebrates and incidental sediment ingestion of 1.8 percent, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day (EPA 1993)

Table 7-49. Raccoon Dose Based on Average Exposure Compared to TRV Values for Upper River

	Fish Tissue average conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww(a)	Dose (:g/gbw/d)(b)	TRV	HQ
Dioxins and furans						
2378-TCDD	0.000000450	na	na	0.00000001	0.0000008	0.01
2378-TCDF	0.000000625	na	na	0.00000001	na	
PCBs						
Total PCB (c)	0.531	0.303123596	0.0987	0.057	0.069	0.83
Pesticides						
Dieldrin	0.008	0.001	0.0003	0.00029	0.015	0.02
Heptachlor Epoxide	0.002	0.001	0.0003	0.00019	0.1	0.0019
Lindane	0.00021	0.0006	0.00015	0.00010	6.15	0.00002
Total DDT	0.08	0.044	0.012	0.008	0.62	0.01
Trace Elements						
Arsenic	0.034	0.938	0.7035	0.155	0.052	**2.99**
Cadmium	0.0047	0.35	0.263	0.058	0.742	0.0779
Lead	0.019	31.2	23.4	5.148	6.15	0.8370
Mercury	0.037	0.058	0.0435	0.010	1	0.01
Methyl mercury	0.037	0.058	0.0435	0.010	0.015	0.68
Selenium	0.14	nd	nd	0.002	0.154	0.02
Zinc	3.93	71	53.3	11.782	123.1	0.10

(a) calculated from dry weight concentrations assuming 85 percent moisture
(b) based on diet of 94 percent fish, 6 percent invertebrates and incidental sediment ingestion of 1.8 percent, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day (EPA 1993)
(c)Total PCB TRV based on Aroclor 1242 in mink

In the lower river zone, HQ values greater than one were calculated for total DDT and methyl mercury in the green heron (Table 7-50) and arsenic, cadmium, lead, mercury, methyl mercury, and zinc in the raccoon (Table 7-51). The largest HQ value was calculated for lead in the raccoon (HQ of 870).

Table 7-50. Green Heron Dose Based on Average Exposure Compared to TRV Values for the Lower River

	Fish Tissue Ave conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww [a]	Dose (:g/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.00000245	na	na	0.0000006	0.000014	0.05
2378-TCDF	0.0000014	na	na	0.0000004	0.000001	0.37
PCBs						
Total PCB	0.703	0.595044248	0.184	0.196	0.41	0.48
Pesticides						
Dieldrin	0.01000	0.000578171	0.00015	0.003	0.077	0.03
Heptachlor Epoxide	0.00580	0.000173451	0.000045	0.002	na	
Lindane	0.000513	0.000	0.00006	0.00014	2	0.0001
Total DDT	0.174	0.058	0.015	0.047	0.003	**15.61**
Trace Elements						
Arsenic	0.09902	1.462	1.1	0.056	2.5	0.02
Cadmium	0.00413	0.292	0.219	0.007	1.45	0.00
Lead	0.04332	38.8	29.1	0.810	1.13	0.72
Mercury	0.078	0.112	0.084	0.023	0.45	0.05
Methyl mercury	0.078	0.112	0.084	0.023	0.0006	**38.06**
Selenium	0.27	0.146	0.11	0.074	0.5	0.15
Zinc	4.56	54.8	41.1	2.328	14.5	0.16

[a] calculated from dry weight concentrations assuming 85 percent moisture

[b] based on diet of 94 percent fish, 6 percent invertebrates and incidental sediment ingestion of 1.8 percent, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day (EPA 1993)

Table 7-51. Raccoon Dose Based on Average Exposure Compared to TRV Values for Lower River

	Fish Tissue Ave. conc. (ug/g ww)	Invertebrates ug/g ww	Sediment ug/g ww [a]	Dose (ug/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.00000245	na	na	0.00000004	0.0000008	0.1
2378-TCDF	0.0000014	na	na	0.00000002	na	
PCBs						
Total PCB [c]	0.703	0.595044248	0.184	0.106	0.069	**1.54**
Pesticides						

Dieldrin	0.01000	0.000578171	0.00015	0.0003	0.015	0.02
Heptachlor Epoxide	0.00580	0.000173451	0.000045	0.0001	0.1	0.001
Lindane	0.000513	0.000	0.00006	0.00005	6.15	0.00001
Total DDT	0.174	0.058	0.015	0.0120	0.62	0.02
Trace Elements						
Arsenic	0.09902	1.462	1.1	0.24	0.052	**4.67**
Cadmium	0.00413	0.292	0.219	0.05	0.742	0.07
Lead	0.04332	38.8	29.1	6.40	6.15	**1.04**
Mercury	0.078	0.112	0.084	0.02	1	0.02
Methyl mercury	0.078	0.112	0.084	0.02	0.015	**1.32**
Selenium	0.27	0.146	0.11	0.03	0.154	0.19
Zinc	4.56	54.8	41.1	9.12	123.1	0.07

[(a)] calculated from dry weight concentrations assuming 85 percent moisture
[(b)] based on diet of 10 percent fish, 90 percent invertebrates and 9.4 percent incidental sediment ingestion, body weight of 5.8 Kg and a calculated ingestion rate of 0.17g/g body weight/day (EPA 1993)
[(c)]Total PCB TRV based on Aroclor 1242 in mink

In the Ship Channel, HQs greater than on were calculated for total PCB and methyl mercury in the green heron (Table 7-52) and total PCBs, lead, and methyl mercury in the raccoon (Table 7-53). The largest HQ value was calculated for methyl mercury in the green heron.

Table 7-52. Green Heron Dose Based on Average Exposure Compared to TRV Values for the Ship Channel

	Fish Tissue [(a)] Ave. conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww[(b)]	Dose (:g/gbw/d)[(c)]	TRV	HQ
Dioxins and furans						
2378-TCDD	na	na	na		0.000014	
2378-TCDF	na	na	na		0.000001	
PCBs						
Total PCB	4.7	0.343	0.121	1.243	0.41	**3.03**
Pesticides						
Dieldrin	0.04	0.001	0.00036	0.011	0.077	0.14
Heptachlor Epoxide	0	0.00046	0.000135	0.000008	na	
Lindane	0	0.0002	0.00006	0.000004	2	0.000002
Total DDT	1.05	0.066	0.01935	0.278	0.003	92.52
Trace Elements						
Arsenic	na				2.5	
Cadmium	na	0.224	0.168	0.005	1.45	0.00
Lead	na	99.8	74.7	2.053	1.13	1.82
Mercury	na	0.158	0.12	0.003	0.45	0.01
Methyl mercury	na	0.158	0.12	0.003	0.0006	**5.43**

Selenium	na	na	na		0.5	
Zinc	na	68.200	51.2	1.404	14.5	0.10

(a) average and maximum fish tissue concentrations are the same (n=1)
(b) calculated from dry weight concentrations assuming 85 percent moisture
(c) based on diet of 94 percent fish, 6 percent invertebrates and incidental sediment ingestion of 1.8 percent, body weight of 212 g and a calculated ingestion rate of 0.28 g/g body weight/day

Table 7-53. Raccoon Dose Based on Average Exposure Compared to TRV Values for Ship Channel

	Fish Tissue (a) Ave. conc. (ug/g ww)	Invertebrates ug/g ww	Sediment ug/g ww(b)	Dose (ug/gbw/d)(c)	TRV	HQ
Dioxins and furans						
2378-TCDD	na	na	na	na	0.0000008	
2378-TCDF	na	na	na	na	na	
PCBs						
Total PCB (d)	4.7	0.343	0.121	0.134	0.069	**1.95**
Pesticides						
Dieldrin	0.04	0.001	0.00036	0.001	0.015	0.06
Heptachlor Epoxide	0	0.00046	0.000135	0.00007	0.1	0.00073
Lindane	0	0.0002	0.00006	0.00003	6.15	0.00001
Total DDT	1.05	0.066	0.01935	0.028	0.62	0.05
Trace Elements						
Arsenic	na				0.052	
Cadmium	na	0.224	0.168	0.037	0.742	0.05
Lead	na	99.8	74.7	16.463	6.15	**2.68**
Mercury	na	0.158	0.12	0.026	1	0.03
Methyl mercury	na	0.158	0.12	0.026	0.015	**1.74**
Selenium	na	na	na		0.154	
Zinc	na	68.200	51.2	11.253	123.1	0.09

(a) average and maximum fish tissue concentrations are the same (n=1)
(b) calculated from dry weight concentrations assuming 85 percent moisture
(c) based on diet of 10 percent fish, 90 percent invertebrates and incidental sediment ingestion of 9.4 percent, body weight of 5.8kg and a calculated ingestion rate of 0.17 g/g body weight/day (EPA 1993)
(d)Total PCB TRV based on Aroclor 1242 in mink

It is important to note that the methyl mercury dose was calculated based on the assumption that 100 percent of the sediment mercury concentration was methyl mercury. This is a conservative estimate of the actual sediment methyl mercury concentrations.

The raccoon diet for the screening level assessment was selected based on the fact that the diet was

dominated by consumption of aquatic prey. Another raccoon diet reported for raccoons living in forested bottomland of Maryland (Llewellyn and Uhler, 1952). This diet contained a much lower contribution from aquatic prey, fish represented 3 percent of the total diet, and aquatic invertebrates represented 37 percent of the total diet. The maximum fish tissue and sediment concentrations were used to estimate the dose due to aquatic prey.

When the dose estimates for the Upper River were compared to the corresponding TRV values only one HQ value exceeded one, arsenic (HQ of 1.86) (Table 7-54). In the Lower River, HQ values greater than one were calculated for total PCBs, arsenic cadmium, lead, mercury, methyl mercury, and zinc (Table 7-55). The largest HQ was calculated for lead (HQ of 1200). Finally, in the Ship Channel, HQ values greater than one were calculated for lead and methyl mercury (Table 7-56).

Table 7-54. Alternate Raccoon Dose Based on Maximum Exposure Compared to TRV Values for Upper River

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww[a]	Dose (:g/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.0000005	na	na	0.000000002	0.0000008	0.003
2378-TCDF	0.0000007	na	na	0.000000004	na	
PCBs						
Total PCB [c]	0.97	0.751	0.2445	0.056	0.069	0.81
Pesticides						
Dieldrin	0.0095	0.003	0.00075	0.00025	0.015	0.02
Heptachlor Epoxide	0.0001	0.002	0.0006	0.00014	0.1	0.0014
Lindane	0.00031	0.001	0.0003	0.00007	6.15	0.00001
Total DDT	0.0901	0.082	0.022	0.006	0.62	0.01
Trace Elements						
Arsenic	0.034	1.292	0.969	0.097	0.052	**1.86**
Cadmium	0.0057	0.524	0.393	0.039	0.742	0.053
Lead	0.104	44.8	33.6	3.355	6.15	0.546
Mercury	0.051	0.118	0.0885	0.009	1	0.01
methyl mercury	0.051	0.118	0.0885	0.009	0.015	0.61
Selenium	0.193	nd	nd	0.001	0.154	0.01
Zinc	5.92	89.4	71.55	6.797	123.1	0.06

[a] calculated from dry weight concentrations assuming 85 percent moisture

[b] based on diet of 3 percent fish, 37 percent invertebrates and incidental sediment ingestion of 9.4 percent, body weight of 5.8kg and a calculated ingestion rate of 0.17 g/g body weight/day

[c] Total PCB TRV based on Aroclor 1242 in mink

Table 7-55. Alternate Raccoon Dose Based on Maximum Exposure Compared to TRV Values for Lower River

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww[a]	Dose (:g/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	0.00001	na	na	0.00000005	0.0000008	0.06
2378-TCDF	0.0000024	na	na	0.00000001	na	
PCBs						
Total PCB [c]	1.27	5.805	1.8	0.400	0.069	**5.80**
Pesticides						
Dieldrin	0.02	0.002	0.000675	0.00024	0.015	0.02
Heptachlor Epoxide	0.017	0.00035	0.00009	0.00011	0.1	0.00
Lindane	0.0012	0.001	0.000135	0.00007	6.15	0.00
Total DDT	0.28	0.004	0.001	0.002	0.62	0.00
Trace Elements						
Arsenic	0.099	5.38	4.04	0.403	0.052	**7.76**
Cadmium	0.0041	0.636	0.477	0.048	0.742	**0.06**
Lead	0.043	115	116	9.087	6.15	**1.48**
Mercury	0.078	0.54	0.405	0.041	1	**0.04**
Methyl mercury	0.078	0.54	0.405	0.041	0.015	**2.72**
Selenium	0.27	0.22	0.165	0.018	0.154	0.12
Zinc	4.56	102.4	76.8	7.691	123.1	**0.06**

[a] calculated from dry weight concentrations assuming 85 percent moisture

[b] based on diet of 3 percent fish, 37 percent invertebrates and 9.4 percent incidental sediment ingestion, body weight of 5.8 kg and a calculated ingestion rate of 0.17 g/g body weight/day (EPA 1993)

[c]Total PCB TRV based on Aroclor 1242 in mink

Table 7-56. Alternate Raccoon Dose Based on Maximum Exposure Compared to TRV Values for Ship Channel

	Fish Tissue Max conc. (:g/g ww)	Invertebrates :g/g ww	Sediment :g/g ww[a]	Dose (:g/gbw/d)[b]	TRV	HQ
Dioxins and furans						
2378-TCDD	na	na	na	na	0.0000008	
2378-TCDF	na	na	na	na	na	
PCBs						
Total PCB [c]	4.7	1.43	0.5025	0.122	0.069	**1.77**
Pesticides						
Dieldrin	0.04	0.003	0.00075	0.0004	0.015	0.03
Heptachlor Epoxide	0	0.002	0.0006	0.0001	0.1	0.0014
Lindane	0	0.001	0.0003	0.0001	6.15	0.00001
Total DDT	1.05	0.082	0.022	0.011	0.62	0.02
Trace Elements						
Arsenic	na		0.969	0.015	0.052	0.30

Cadmium	na	0.662	0.393	0.048	0.742	0.06
Lead	na	726.000	33.6	46.202	6.15	**7.51**
Mercury	na	1.843	0.0885	0.117	1	0.12
Methyl mercury	na	1.843	0.0885	0.117	0.015	**7.82**
Selenium	na	na	na		0.154	
Zinc	na	218.000	71.55	14.856	123.1	0.12

[a] calculated from dry weight concentrations assuming 85 percent moisture
[b] based on diet of 3 percent fish, 37 percent invertebrates and 9.4 percent incidental sediment ingestion, body weight of 5.8 Kg and a calculated ingestion rate of 0.17 g/g body weight/day (EPA 1993)
[c]Total PCB TRV based on Aroclor 1242 in mink

REFERENCES FOR CHAPTER 7

ACOE. 1988. Environmental effects of dredging. Technical notes. Relationship between PCB tissue residues and reproductive success of fathead minnows. EEDP-01-13. U.S. Army Corps of Engineers, Engineer Waterways Experiment Station, Environmental Laboratory, Vicksburg, MS.

Adams, W.J., G.M. DeGraeve, T.D. Sabourin, J.D. Cooney, and G.M. Mosher. 1986. Toxicity and bioconcentration of 2,3,7,8-TCDD to fathead minnows (Pimephales promelas). Chemosphere 15:1503-1511.

Allen, H.E., G. Fu, and B. Deng. 1993. Analysis of acid-volatile sulfide and simultaneously extracted metals for the estimation of potential toxicity in aquatic sediments. Environ. Toxicol. Chem. 12:1441-1453.

Ankley, G.T., D.E Tillitt, J.P. Giesy, P.D. Jones, and D.A. Berbrugge. 1991. Bioassay derived 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents in PCB-containing extracts from flesh and eggs of Lake Michigan chinook salmon (Oncorhynchus tshawytscha) and possible implications for reproduction. Can. J. Fish. Aquat. Sci. 48:1685-1690.

Ankley, G.T., V.R. Mattson, E.N. Leonard, C.W. West, and J.L. Bennett. 1993. Predicting the acute toxicity of copper in freshwater sediments: Evaluation of the role of acid-volatile sulfide. Environ. Toxicol. Chem. 12:315-320.

ATSDR. 1992. Toxicological profile for 4,4'-DDT, 4,4'-DDE, and 4,4'-DDD (draft). U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA. 131 pp. + appendices.

Baumann, P.C. 1984. Cancer in wild freshwater fish populations with emphasis on the Great Lakes. J. Great Lakes Res. 10:251-253.

Baumann, P.C., W.D. Smith, and M. Riluck. 1982. Hepatic tumor rates and polynuclear aromatic hydrocarbon levels in two populations of brown bullhead (*Ictalurus nebulosus*). In: Polynuclear Aromatic Hydrocarbons, Physical and Biological Chemistry, Sixth International Symposium.

Baumann, P.C., M.J. Mac, S.B. Smith, and J.C. Harshbarger. 1991. Tumor frequencies in walleye (*Stizostedion vitreum*) and brown bullhead (*Ictalurus nebulosus*) and sediment contaminants in tributaries of the Laurentian Great Lakes. Can. J. Fish Aquat. Sci. 48:1804-1810.

Baumann, P.C., I.R. Smith, and C.D. Metcalfe. 1996. Linkages between chemical contaminants and tumors in benthic Great Lakes fish. J. Great Lakes Res. 22(2):131-152.

Biesinger, K.E., L.E. Anderson, and J.G. Eaton. 1982. Chronic effects of inorganic and organic mercury on *Daphnia magna*: Toxicity, accumulation, and loss. Arch. Environ. Contam. Toxicol. 11:769-774.

Bennett, W.N., A.S. Brooks, and M.E. Borass. 1986. Selenium uptake and transfer in an aquatic food chain and its effects on fathead minnow larvae. Arch. Environ. Contam. Toxicol. 15:513-517.

Benoit, D.A., E.N. Leonard, G.M. Christensen and J.T. Fiandt. 1976. Toxic effects of cadmium on three generations of brook trout (*Salvelinus fontinalis*). Trans. Am. Fish. Soc. 105:550-560.

Berlin, W.H., R.J. Hesselberg, and M.J. Mac. 1981. Growth and mortality of fry of Lake Michigan lake trout during chronic exposure to PCBs and DDE. Tech. Pap. US Fish Wildl. Ser. 105:11-22.

Birge, W.J., J.A. Black, A.G. Westerman, and J.E. Hudson. 1979. The effects of mercury on reproduction of fish and amphibians. In: The biogeochemistry of mercury in the environment. Nriagu (ed). Elsevier/North Holland Biomedical Press, Amsterdam. pp. 629-655.

Black, J.J., P.P. Dymerski and W.F. Zapisek. 1980. Fish tumor pathology and aromatic hydrocarbons pollution in a Great Lakes estuary. In: Hydrocarbons and Halogenated Hydrocarbons in the Aquatic Environment. B.K. Afghan

and D. Markay (eds). Plenum Press.

Branson, D.R., G.E. Blau, H.C. Alexander, and W.B. Neely. 1975. Bioconcentration of 2,2',4, 4'-tetrachlorobiphenyl in rainbow trout as measured by an accelerated test. Trans. Am. Fish. Soc. 104:785-792.

Brown, D.A., C.A. Bawden, K.W. Chatel, and T.R. Parsons. 1970. The wildlife community of Iona Island jetty, Vancouver, B.C., and heavy-metals pollution effects. Environ. Conserv. 4:213-216.

Brunström, B. and P.O. Darnerud. 1983. Toxicity and distribution in chick embryos of 3,3',4,4'-tetrachlorobiphenyl injected into the eggs. Toxicology 27:103-110.

Brunström, B. 1988. Sensitivity of embryos from duck, goose, herring gull, and various chicken breeds to 3,3′,4,4′-tetrachlorobiphenyl. Poultry Sci. 67:52-57.

Brunström, B. 1990. Mono-ortho-chlorinated chlorobiphenyls: Toxicity and induction of 7-ethoxyresorufin O-deethylase (EROD) activity in chick embryos. Arch. Toxicol. 64:188-192.

Buchman, M. 1999. NOAA Screening Quick Reference Tables. HAZMAT Report 99-1. NOAA Coastal Protection and Restoration Division, Seattle, WA. pp. Available at response restoration.noaa.gov/cpr/sediment/squirt/squirt.html.

Burdick, G.E., E.J. Harris, H.J. Dean, T.M. Walker, J. Skea, and D. Colby. 1964. The accumulation of DDT in lake trout and the effect on reproduction. Trans. Amer. Fish. Soc. 93:127-136.

Burnett, K.M. and W.J. Liss. 1990. Multi-steady-state toxicant fate and effect in laboratory aquatic ecosystems. Environ. Toxicol. Chem. 9:637-647.

Burton, Jr. G.A. 1991. Assessing the toxicity of freshwater sediments. Env. Tox. Chem. 10:1585-1627.

Carey, J.H. 1994. Transformation processes of contaminants in rivers. Hydrological, chemical and biological processes of transformation and transport of contaminants in aquatic environments. Proceedings of the Rostov-on-Don Symposium;1993 May. Rostov, Russia: International Association of Hydrological Sciences (IAHS) Pub. No 219. p41-50.

Carey, J., P. Cook, J. Giesy, P. Hodson, D. Muir, W. Owens, and K. Solomon. 1998. Ecotoxicological risk assessment of the chlorinated organic chemicals. SETAC Press, Pensacola, FL.

Carlson, A.R., G.L. Phipps, V.R. Mattson, P.A. Kosian, and A.M. Cotter. 1991. The role of acid-volatile sulfide in determining cadmium bioavailability and toxicity in freshwater sediments. Environ. Toxicol. Chem. 10:1309-1319.

Cearley, J.E. and R.L. Coleman. 1974. Cadmium toxicity and bioconcentration in largemouth bass and bluegill. Bull. Environ. Contam. Toxicol. 11:146-151.

Chen, T.T., P.C. Reid, V. Van Beneden, and R.A. Sonstegard. 1986. Effect of Aroclor 1254 and mirex on estradiol-induced vitellogenin production in juvenile rainbow trout (*Salmo gairdeneri*). Can. J. Fish. Aquat. Sci. 43:169-173.

Clement Associates. 1985. Chemical, physical, and biological properties of compounds present at hazardous waste sites. Prepared for the U.S. Environmental Protection Agency, Washington, DC, by Clement Associates.

Cleveland, L., E.E. Little, D.R. Buckler, and R.H. Wiedmeyer. 1993. Toxicity and bioaccumulation of waterborne and dietary selenium in juvenile bluegill (*Lepomis macrochirus*). Aquat. Toxicol. 27:265-280.

Couture, L.A., B.D. Abbot, and L.S. Birnbaum. 1990. A critical review of the developmental toxicity and teratogenicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin: recent advances toward understanding the mechanism. Teratology 42:619-627.

Cuerrier, J.P. J.A. Keith, and E. Stone. 1967. Problems with DDT in fish culture operations. Naturaliste Can. 94:315-320.

Di Toro, D.M., J.D. Mahony, D.J. Hansen, K.J. Scott, M.B. Hicks, S.M. Mayr, and M.S. Redmond. 1990. Toxicity of cadmium in sediments: The role of acid-volatile sulfide. Environ. Toxicol. Chem. 9:1487-1502.

Di Toro, D.M., J.D. Mahony, D.J. Hansen, K.J. Scott, A.R. Carlson, and G.T. Ankley. 1992. Acid-volatile sulfide predicts the acute toxicity of cadmium and nickel in sediments. Environ. Sci. Technol. 26:96-101.

Eisler, R. 1985. Cadmium hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.2). U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD. 46 pp.

Eisler, R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: A synoptic review. Biological report 85(1.7). U.S. Department of the Interior, Fish and Wildlife Service. Washington, DC. 72 pp.

Eisler, R. 1987. Polycyclic aromatic hydrocarbon hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.11). U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD. 81 pp.

Eisler, R. 1988a. Arsenic hazards to fish, wildlife, and invertebrates: A synoptic review. Biological Report 85(1.12). U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD. 92 pp.

Eisler, R. 1988b. Lead hazards to fish, wildlife and invertebrates: A synoptic review. Biological Report 85(1.14). U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD. 134 pp.

Environmental Health Data Search. no date. (p. 19)

ESI. 1998. Review of tissue residue effects data for tributyltin, mercury, and polychlorinated biphenyls. Draft. Prepared for Port of Seattle, Lockheed Martin Corporation, and Todd Shipyards. EVS Solutions, Inc., Seattle, WA.

EXTOXNET. 1996. Pesticide information profiles, Extension Toxicology Network.

Fabacher, D.L., J.M. Besser, C.J. Schmitt, J.C. Harshbarger, P.H. Peterman, and J.A. Lebo. 1991. Contaminated sediments from tributaries of the Great Lakes: Chemical characterization and carcinogenic effects in medaka (*Oryzias latipes*). Arch. Environ. Contam. Toxicol. 20:17-34.

Firestone, D. 1973. Etiology of chick edema disease. Environ. Health Perspect. 5:59-66.

Fisher, J.P, J.M. Spitsbergen, B. Bush, and B. Jahan-Parwar. 1994. Effect of embryonic PCB exposure on hatching success, survival, growth, and developmental behavior in landlocked Atlantic salmon, *Salmo salar*. In: Environmental toxicology and risk assessment: 2nd Volume . ASTM STP 1216. J.W. Gorsuch, F.J. Dwyer, C.G. Ingersoll, and T.W. LaPoint (eds). American Society for Testing and Materials, Philadelphia PA. pp. 298-314.

Folmar, L.C., W.W. Dickhoff, W.S. Zaugg, and H.O. Hodgins. 1982. The effects of Aroclor 1254 and No. 2 fuel oil on smoltification and sea-water adaptation of coho salmon (*Oncorhynchus kisutch*). Aquat. Toxicol. 2:291-299.

Friedmann, A.S., M.C. Watzin, T. Brinck-Johnsen, and J.C. Leiter. 1996. Low levels of dietary methylmercury inhibit growth and gonadal development in juvenile walleye (*Stizostedion vitreum*). Aquatic Tox. 35:265-278.

Giesy, J.P., J.P. Ludwig, and D.E. Tillitt. 1994a. Dioxins, dibenzofurans, PCBs and colonial, fish-eating water birds. In: Dioxins and health. A. Schecter (ed). Plenum Press, New York, NY.

Giesy, J.P., J.P. Ludwig, and D.E. Tillitt. 1994b. Deformities in birds of the Great Lakes region, assigning causality. Environ. Sci. Technol. 28(3):128-135.

Gilani, S.H. and M. Morano. 1979. Chromium poisoning and chick embryogenesis. Environ. Res. 19:427-431.

Gissel-Nielsen, M. and G. Gissel-Nielsen. 1978. Sensitivity of trout to chronic and acute exposure to selenium. Agric. Environ. 4:85-91.

Hamilton, S.J., P.M. Mehrle, and J.R. Jones. 1987a. Cadmium-saturation technique for measuring metallothionein in brook trout. Trans. Am. Fish. Soc. 116:541-550.

Hamilton, S.J., P.M. Mehrle, and J.R. Jones. 1987b. Evaluation of metallothionein measurement as a biological indicator of stress from cadmium in brook trout. Trans. Am. Fish. Soc. 116:551-560.

Hamilton, S.J., K.J. Buhl, N.L. Faerber, R.H. Widmeyer, and F.A. Bullard. 1990. Toxicity of organic selenium in the diet of chinook salmon. Environ. Toxicol. Chem. 9:347-358.

Hansen, L.G., W.B. Wiekhorst, and J. Simon. 1976. Effects of dietary Aroclor 1242 on channel catfish (*Ictalurus punctatus*) and the selective accumulation of PCB components. J. Fish. Res. Bd. Can. 33:1343-1352.

Hawryshyn, C.W. and W.C. Mackay. 1979. Toxicity and tissue uptake of methylmercury administered intraperitoneally to rainbow trout (*Salmo gairdneri* Richardson). Bull. Environm. Contam. Toxicol. 23:79-86.

Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. Dietary exposure of mink to carp from Saginaw Bay, Michigan. Effects on reproduction and survival, and the potential risks to wild mink populations. Arch. Environ. Contam. Toxicol. 28:334-343.

Hendricks, J.D., W.T. Scott, T.P. Putnam, and R.O. Sinnhuber. 1981. Enhancement of aflatoxin B1 hepatocarcinogenesis in rainbow trout (*Salmo gairdneri*) embryos by prior exposure of gravid females to dietary Aroclor 1254. In: Aquatic Toxicology and Hazard Assessment. Fourth Conference. D.R. Branson and K.L. Dickson (eds). ASTM STP 737. American Society of Testing and Materials, Philadelphia, PA. pp. 203-214.

Herson-Jones, L., A. Warner, B. Jordan, and K. Hagan. 1994. Anacostia Watershed water quality report: 1987-1990. Metropolitan Washington Council of Governments. Prepared for the District of Columbia Department of Consumer and Regulatory Affairs. Washington D.C.

Hoffman, D.J. and M.L. Gay. 1981. Embryotoxic effects of benzo(a)pyrene, chrysene, and 7,12-dimethylbenz(a)anthracene in petroleum hydrocarbon mixtures in mallard ducks. J. Toxicol. Environ. Health 7:775-787.

Hoffman, D.J. and J.M. Moore. 1979. Teratogenic effects of external egg applications of methyl mercury in the mallard, *Anas platyrhynchos*. Teratology 20:453-462.

Hogan, J.W. and J.L. Brauhn. 1975. Abnormal rainbow trout fry from eggs containing high residues of a PCB (Aroclor 1242). The Progressive Fish-Culturist 37(4):229-230.

Holcombe, G.W., D.A. Benoit, and E.N. Leonard. 1976. Long-term effects of lead exposure on three generations of brook trout (*Salvelinus fontinalis*). J. Fish. Res. Bd. Can. 33:1731-1741.

Horness, B.H., D.P. Lomax, L.L. Johnson, M.S. Myers, S.M. Pierce, and T.K. Collier. 1998. Sediment quality thresholds: Estimates from hockey stick regression of liver lesion prevalence in English sole (*Pleuronectes vetulis*). Environ. Toxicol. Chem. 17(5): 872-882.

Jarvinen, A.W., M.J. Hoffman, and T.W. Thorslund. 1977. Long-term toxic effects of DDT food and water exposure on fathead minnows (*Pimephales promelas*). J. Fish. Res. Board Can. 34:2089-2103.

Jarvinen, A.W. and G.T. Ankley. 1998.

Jarvinen, A.W. and G.T. Ankley. 1999. Linkage of effects to tissue residues: Development of a comprehensive database for aquatic organisms exposed to inorganic and organic chemicals. SETAC Press. Pensacola, FL.

Johnson, L.L., E. Casillas, T.K. Collier, B.B. McCain, and U. Varanasi. 1988. Contaminant effects on ovarian maturation in English sole (*Parophrys vetulus*) from Puget Sound, Washington. Can. J. Fish. Aquat. Sci. 45:2133-2146.

Jones, P.D., J.P. Giesy, J.L. Newsted, D.A. Verbrugge, D.L Beaver, G.T. Ankley, D.E. Tillitt, K.B. Lodge, and G.J.

Niemi. 1993. 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents in tissues of birds at Green Bay Wisconsin, USA. Arch. Environ. Contam. Toxicol. 24:345-354.

Jones, P.D., J.P. Giesy, J.L. Newsted, D.A. Verbrugge, J.P. Ludwig, M.E. Ludwig, H.J. Auman, R. Crawford, D.E. Tillitt, T.J. Kubiak, and D.A. Best. 1994. Accumulation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents in double-crested cormorant chicks in the North American Great Lakes. Ecotoxicol. Environ. Safety 27:192-209.

Kleeman, J.M. J.R. Olson, S.M. Chen, and R.E. Peterson. 1986. Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in rainbow trout. Toxicol. Appl. Pharmacol. 83:391-401.

Kumada, H. S. Kimura, M. Yokote, and Y. Matida. 1973. Acute and chronic toxicity, uptake and retention of cadmium in freshwater organisms. Bull. Freshwater Fish. Res. Lab (Tokyo). 22:157-165.

Landrum, P.F., B.J. Eadie, and W. R. Faust. 1991. Toxicokinetics and toxicity of a mixture of sediment-associated polycyclic aromatic hydrocarbons to the amphipod *Diporeia* sp. Env. Tox. Chem. 10:35-46.

Leland, H.V. and J.S. Kuwabara. 1985. Trace metals. In: Fundamentals of aquatic toxicology: Methods and applications. G.M. Rand and S.R. Petrocelli (eds). Hemisphere Publishing Corporation, New York, NY. 666 pp.

Leonards, P.E.G., T.H. de Vries, W. Minnard, S. Stuifzand, W.P. de Vooght, W.P. Cofino, N.M. Straalen, and B. Hattum. 1995. Assessment of experimental data on PCB-induced reproduction inhibition in mink, based on an isomer- and congener-specific approach using 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxic equivalency. Environ. Toxicol. Chem. 14(4):639-652.

Lipnick, R.L. 1993. Baseline toxicity QSAR models: A means to assess mechanism of toxicity for aquatic organisms and mammals. pp. 610-619. In: Volume 2, Environmental toxicology and risk assessment. J.W. Gorsuch, F.J. Dwyer, C.G. Ingersoll, and T.W. Lapoint (eds). STP 1216. American Society for Testing and Materials. Philadelphia, PA.

Lippson et al. 1980. Environmental Atlas of the Potomac Esturary. Environmental Center, Martin Maerietta Corp. Prepared for Power Plant Siting Program, Maryland Department of Natural Resources. pp. 280.

Llewellyn, L.M. and F.M. Uhler. 1952. The foods of fur animals of the Patuxent Research Refuge, Maryland. Am. Midl. Nat. 48:193-203.

Mac, M.J. and C.C. Edsall. 1991. Environmental contaminants and the reproductive success of lake trout in the Great Lakes: An epidemiological approach. J. Toxicol. Environ. Health 33:375-394.

Mac, M.J. and T.R. Schwartz. 1992. Investigations into the effects of PCB congeners on reproduction in lake trout from the Great Lakes. Chemosphere 25:189-192.

Macek, K.J., K.S. Buxton, S.K. Derr, J.W. Dean, and S. Sauter. 1976. Chronic toxicity of lindane to selected aquatic invertebrates and fish. EPA 600/3-76-046. U.S. Environmental Protection Agency, Office of Research and Development, Duluth, MN. 50 pp.

Macek, K.J. 1968. Reproduction in brook trout (*Salvelinus fontinalis*) fed sublethal concentrations of DDT. J. Fish. Res. Bd. Can. 25:1787-1796.
Malins, D.C., B.B. McCain, M.S. Myers, D.W. Brown, M.M. Krahn, W.T. Roubal, M.H. Schiewe, J.T. Landahl, and S.L. Chan. 1987. Field and laboratory studies of the etiology of liver neoplasms in marine fish from Puget Sound. Environ. Health Perspectives 71:5-16.

Marco, A., C. Quilchano, and A.R. Blaustein. 1999. Sensitivity to nitrate and nitrite in pond-breeding amphibians from the Pacific Northwest, USA. Env. Tox. Chem. 18(12):2836-2839.

Mayer, F.L. and M.R. Ellersieck. 1986. Manual of acute toxicity: Interpretation and data base for 401 chemicals and 66 species of freshwater animals. Resource Publication 160. U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, MD.

McCarty, L.S. and Mackay, D. 1993. Enhancing ecotoxicological modeling and assessment: Body residues and modes of toxic action. Environ. Sci. Technol. 27:1719-1728.

McGeachy, S.M. and D.G. Dixon. 1990. Effect of temperature on the chronic toxicity of arsenate to rainbow trout (*Oncorhynchus mykiss*). Can. J. Fish. Aquat. Sci. 47:2228-2234.

Meador, J.P., J.E. Stein, W.L. Reichert, and U. Varanasi. 1995. Bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. Rev. Env. Cont. Tox. 143.

Mehrle, P.M. D.R. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. DeGraeve, J.J. Coyle, and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout. Environ. Toxicol. Chem. 7:47-62.

Miller, R.A., L. A. Norris, and B.R. Lopez. 1979. The response of Coho salmon and guppies to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in water. Trans. Am. Fish. Soc. 108:401-407.

Monosson, E. 1999. Reproductive, developmental, and immunotoxic effects of PCBs in fish: a summary of laboratory and field studies. Prepared for National Oceanic and Atmospheric Administration Damage Assessment Center, Silver Spring, MD. Prepared through Industrial Economics, Inc., Cambridge, MA.

Moore, J.W. and S. Ramamoorthy. 1984. Organic chemicals in natural water. Springer-Verlag, New York, NY.

National Park Service (NPS). 2000. National Park Service, Anacostia Park. A Unit of National Parks East. http://www.nps.gov/nace/anacostia.htm

Nebeker, A. 1988. Personal communication (telephone call on February 26, 1988 to J. Field, National Oceanic and Atmospheric Administration, Seattle, WA). U.S. Environmental Protection Agency, Corvallis, OR.

Neff, J.M. 1979. Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fates, and biological effects. Applied Science Publishers, London.

Niimi, A.J. 1996. PCBs in aquatic organisms. In: Environmental contaminants in wildlife: Interpreting tissue concentrations. W.N Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds). Society of Environmental Toxicology and Chemistry. CRC Press, Boca Raton, FL. pp. 117-152.

NOAA. 1999. Screening quick reference table. U.S. Department of Commerce, National Oceanic and Atmospheric Administration, Hazardous Materials Response Branch, Seattle, WA. 12 pp.

Ogle, R.S. and A.W. Knight. 1989. Effect of elevated foodborne selenium on growth and reproduction of the fathead minnow (*Pimephales promelas*). Arch. Environ. Contam. Toxicol. 18:795-803.

O'Connor, J.M. and R.J. Huggett. 1988. Aquatic pollution problems, North Atlantic coast, including Chesapeake Bay. Aquat. Toxicol. 11:163-190.

Pascoe, D., S.A. Evans, and J. Woodworth. 1986. Heavy metal toxicity to fish and the influence of water hardness. Arch. Environ. Contam. Toxicol. 15:481-487.

Patton, J.F. and M.P. Dieter. 1980. Effects of petroleum hydrocarbons on hepatic function in the duck. Comp. Biochem. Physiol. 65C:33-36.

Peterson, R.E. and M.K. Walker. 1992. Effects on aquatic and terrestrial organisms. Current views on the impact of dioxins and furans on human health and the environment. Session V: Ecotoxicological aspects. pp. 383-399. In: Proceedings of the Toxicology Forum, November 9-11, 1992, Berlin, Germany.

Peterson, R.E., H.M. Theobald, and G.L. Kimmel. 1993. Developmental and reproductive toxicity of dioxins and related compounds: cross-species comparisons. CRC Crit. Rev. Toxicol. 23:283-335.

Pinkney, A.E., J.C. Harshbarger, E.B. May, and M.J. Melancon. 2000. Tumor prevalences in brown bullheads

(*Ameirus nebulosus*) from the tidal Potomac River Watershed and biomarkers of exposure and response. Report CBFO-C99-04. U.S. Fish and Wildlife Service, Annapolis, MD.

Ridgeway, L.P. and D.A. Karnofsky. 1952. The effects of metals on the chick embryo: Toxicity and production of abnormalities in development. Ann. N.Y. Acad. Sci. 55:203-215.

Sample, B.E., D.M. Opresko, and G.W. Suter II. 1996. Toxicological benchmarks for wildlife: 1996 revision. Prepared by Oak Ridge National Laboratory, Risk Assessment Program, Health Sciences Research Division, Oak Ridge, TN.

Sanderson, J.T., J.E. Elliott, R.J. Norstrom, P.E. Whitehead, L.E. Hart, K.M. Cheng, and G.D. Bellward. 1994. Monitoring biological effect of polychloroinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in great blue heron chicks in British Columbia. Toxicol. Environ. Health. 41:435-450.

Sanderson, J.T., D.M. Janz, G.D. Bellward, and J.P. Giesy. 1997. Effects of embryonic and adult exposure to 2,3,7,8-tetrachlorofibenzo-*p*-dioxin in hepatic microsomal testosterone hydroxylase activities in great blue herons. Environ. Toxicol. Chem. 16(6):1304-1310.

Schimmel, S.C., J.M. Patrick, and J. Forester. 1976. Heptachlor: Toxicity to and uptake by several estuarine organisms. J. Toxicol. Environ. Health 1:955-965

Schrankel, K.R., B.L. Kreamer, and M.T.S. Hsia. 1982. Embryotoxicity of 3,3', 4,4'-tetrachloroazoxybenzene in the chick embryo. Arch. Environ. Contam. Toxicol. 11:195-202.

Scott, W.B. and E.J. Crossman. 1973. Freshwater fishes of Canada. Department of Fisheries and Oceans, Fisheries Research Board of Canada, Ottawa. Bulletin 184. 966 pp.

Sivarajah, K., C.S. Franklin, and W.P. Williams. 1978. The effects of polychlorinated biphenyls on plasma steroid level and hepatic microsomal enzymes in fish. J. Fish Biol. 13:401-409.

Slaga, T.J., W.M. Bracken, A. Viaje, D.L. Berry, S.M. Fischer, D.R. Miller, W. Levin, A.H. Convey, H. Yagi, and D.M. Jerina. 1978. Tumor initiating and promoting activities of various benzo(a)pyrene metabolites in mouse skin. pp. 371-392. In: P.W. Jones and R.I. Freudenthal (eds.), Carcinogenesis. A comprehensive survey. Vol. 3. Polynuclear aromatic hydrocarbons: Second international symposium on analysis, chemistry, and biology. Raven Press, New York, NY.

Smith, S.L., D.D. MacDonald, K.A. Keenleyside, C.G. Ingersoll, and L.J. Field. A Preliminary Evaluation of Sediment Quality Assessment Values for Freshwater Ecosystems. J. Great Lakes Res. 22(3):624-638.

Snarski, V.M. and G.F. Olson. 1982. Chronic toxicity and bioaccumulation of mercuric chloride in the fathead minnow (*Pimephales promelas*). Aquatic Toxicol. 2:143-156.

Sorensen, E.M.B. 1976. Toxicity and accumulation of arsenic in green sunfish, *Lepomis cyanellus*, exposed to arsenate in water. Bull. Environ. Contam. Toxicol. 15:756-761.

Spehar, R.L. 1976. Cadmium and zinc toxicity to flagfish (*Jordanella floridae*). J. Fish. Res. Bd. Can. 33:1939-1945.

Spies, R.B., D.W. Rice, Jr., P.A. Montagna and R.R. Ireland. 1985. Reproductive success, xenobiotic contaminants, and hepatic mixed function oxidase (MFO) activity in *Platichthys stellatus* populations from San Francisco Bay. Mar. Environ. Res. 17:117-121.

Spitzbergen, J.M., M.K. Walker, J.R. Olson and R.E. Peterson. 1991. Pathologic alternations in early life stages of lake trout, *Salvelinus namaycush*, exposed to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin as fertilized eggs. Aquat. Toxicol. 19:41-72.

Summer, C., J. Giesy, S. Bursian, J. Render, T. Kubiak, P. Jones, D. Verbrugge, and R. Aulerich. 1996a. Effects induced by feeding organochlorine-contaminated carp from Saginaw Bay, Lake Huron, to laying white leghorn

hens. I. Effects of health of adult hens, egg production, and fertility. J. Toxicol. Environ. Health. 49:389-407.

Summer, C., J. Giesy, S. Bursian, J. Render, T. Kubiak, P. Jones, D. Verbrugge, and R. Aulerich. 1996b. Effects induced by feeding organochlorine-contaminated carp from Saginaw Bay, Lake Huron, to laying white leghorn hens. II. Embryotoxic and teratogenic effects. J. Toxicol. Environ. Health. 49:409-438.

Suter, G.W., L.W. Barnthouse, R.A. Efroymson, and H. Jager. 1999. Ecological risk assessment in a large river-reservoir: 2. Fish community. Envir. Toxicol. Chem. 18(4):589-598.

Thomas, P. 1988. Reproductive endocrine function in female Atlantic croaker exposed to pollutants. Mar. Environ. Res. 24:179-183.

Tillitt, D.E., G.T. Ankley, J.P. Giesy, J.P. Ludwig, H.K. Matsuba, D.V. Weseloh, P.S. Ross, C.A. Bishop, L. Sileo, K.L. Stromberg, J. Larson, and T.J. Kubiak. 1992. Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes. Environ. Toxicol. Chem. 11:1281-1288.

Tracey, G.A. and D.J. Hansen. 1996. Use of biota-sediment accumulation factors to assess similarity of nonionic organic chemical exposure to benthically-coupled organisms of differing trophic mode. Arch. Environ. Contam. Toxicol. 30:467-475.

Udvardy, M.D.F. and J. Farrand. 1994. National Audubon Society field guide to North American birds. Western Region. Alfred A. Knopf, New York.

U.S. EPA. 1980. Ambient water quality criteria for polynuclear aromatic hydrocarbons. U.S. Environmental Protection Agency Report 440/5-80-069. 193 pp.

U.S. EPA. 1986. Quality criteria for water 1986. U.S. Environmental Protection Agency, Office of Water, Washington, DC. 456 pp.

U.S. EPA. 1990. Macroinvertebrate field and laboratory methods for evaluating the biological integrity of surface waters. Report No. 600/4-90/030. Office of Research and Development, U.S. Environmental Protection Agency, Cincinnati, OH.

U.S. EPA. 1992. A framework for ecological risk assessment. EPA 630-R-92-001. U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC.

U.S. EPA. 1993. Wildlife exposure factors handbook. EPA/600/R-93/187a. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC.

U.S. EPA. 1994. Estimating exposure to dioxin-like compounds. Volumes I, II, and III. EPA /600/6-88/005Ca. External review draft. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC.

U.S. EPA. 1996. Assessment and remediation of contaminated sediments (ARCS) program: calculation and evaluation of sediment effect concentrations for the amphipod *Hyelella azteca* and the midge *Chironomus riparius*. EPA 905-R96-008. U.S. Environmental Protection Agency, Great Lakes National Program Office, Chicago, IL.

U.S. EPA. 1997. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments, interim final. EPA 540-R-97-006. U.S. Environmental Protection Agency, Environmental Response Team, Edison, NJ.

USFWS. 1997. Data report: determination of toxicity and concentration of inorganic and organic contaminants in sediments used to restore Kenilworth Marsh, Washington DC. Publication No. CFBO-D97-02. U.S. Fish and Wildlife Service, Annapolis, MD.

Van Luik, A. 1984. Mined land reclamation using polluted urban navigable waterway sediments. II: Organics. J. Environ. Qual. 13(3):415-422.

Varanasi, U., J.E. Stein, and M. Nishimoto. 1989. Biotransformation and disposition of polycyclic aromatic hydrocarbons (PAH) in fish. In: Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment. U. Varanasi (ed). CRC Press Inc., Boca Raton, FL. 93 pp.

Varanasi, U., J.E. Stein, L.L. Johnson, T.K. Collier, E. Casillas, and M.S. Myers. 1992. Evaluation of bioindicators of contaminant exposure and effects in coastal ecosystems. In: Ecological indicators, Vol. 1. D.H. McKenzie, D.E. Hyatt, and V.J. McDonald (eds). Proceedings of an international symposium, Ft. Lauderdale, FL.

Velinsky, D.J., C. Haywood, T.L. Wade, E. Reinharz. 1992. Sediment contamination studies of the Potomac and Anacostia Rivers around the District of Columbia. Interstate Commission on the Potomac River Basin. ICPRB Report No. 92-2. Submitted to the District of Columbia Department of Consumer and Regulatory Affairs. Washington D.C.

Velinsky, D.J., G.F. Riedel, and G. Foster. 1999. Effects of stormwater runoff on the water quality of the Tidal Anacostia River. Prepared for U.S. EPA-Region III, Water Protection Division.

von Westernhagen, H., H. Rosenthal, V. Dethlefsen, W. Ernst, U. Harms, and P.D. Hansen. 1981. Bioaccumulating substances and reproductive success in Baltic flounder *Platichthys flesus*. Aquatic Tox. 1:85-99.

Walker, M.K. and R.E. Peterson. 1991. Potencies of polychlorinated dibenzo-*p*-dioxin, dibenzofuran, and biphenyl congeners, relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, for producing early life stage mortality in rainbow trout (*Oncorhynchus mykiss*). Aquat. Toxicol. 21:219-238.

Walker, M.K., J.M. Spitsbergen, J.R. Olson, and R.E. Peterson. 1991. 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) toxicity during early life stage development of lake trout (*Salvelinus namaycush*). Can. J. Fish. Aquat. Sci. 48:875-883.

Ware, G.W. 1997. An introduction to insecticides. University of Minnesota National IPM Network. (http://ipmworld.umn.edu/chapter/ware.htm)

Weeks, B.A. and J.E. Warinner. 1984. Effects of toxic chemicals on macrophage phagocytosis in two estuarine fishes. Mar. Environ. Res. 14:327-335.

Weeks, B.A. and J.E. Warinner. 1986. Functional evaluation of macrophages in fish from a polluted estuary. Vet. Immunol. Immunopathol. 12:313-320.

Weeks, B.A., J.E. Warinner, P.L. Mason, and D.S. McGinnis. 1986. Influence of toxic chemicals on the chemotactic response of fish macrophages. J. Fish Biol. 28:653-658.

Wiener, J.G. and D.J. Spry. 1996. Toxicological significance of mercury in freshwater fish. pp. 297-339. In: Environmental Contaminants in Wildlife. W.N. Beyer, G.H. Heinz, and A.W. Redmon-Norwood (eds). Lewis Publisher, Boca Raton, FL.

Wisk, J.D. and K.R. Cooper. 1990. Comparison of the toxicity of several polychlorinated dibenzo-*p*-dioxins and 2,3,7,8-tetrachlorodibenzofuran in embryos of the Japanese medaka (*Oryzias ladipes*). Chemosphere 20:361-377.

Wren, C.D. 1991. Cause-effect linkages between chemicals and populations of mink (*Mustela vison*) and otter (*Lutra canadensis*) in the Great Lakes Basin. J. Toxicol. Environ. Health. 33:549-585.

Zabel, E.W., P.M. Cook, and R.E. Peterson. 1995. Toxic equivalency factors of polychlorinated dibenzo-*p*-dioxin, dibenzofuran and biphenyl congeners based on early life stage mortality in rainbow trout (*Oncorhynchus mykiss*). Aquat. Toxicol. 31:315-328.

8. CONCLUSION AND SUMMARY

8.1 CONCEPTUAL SITE MODEL

A preliminary conceptual site model (CSM) of the tidal Anacostia River was developed that identifies the major pathways and receptors that may contact the river (Figure 6-1). A CSM describes the processes that link sources of contamination at a site to exposures of human or ecological receptors. Ideally, the model defines the inputs of constituents to a site, the physical and chemical processes that result in transport of the constituents into environmental media to which human or ecological receptors may come into contact, and identifies the receptors that are likely to be impacted by exposure to these media. In its mature form, the model provides a basis for planning of data collection and evaluation needed to support risk assessments and remedial actions. In the early stages of development, the CSM identifies all *potential* links between sources and receptors, which subsequently can be evaluated for their plausibility and relevance with further data collection and analysis. Inputs, fate and transport processes, and exposure scenarios that are subsequently determined to be implausible or of negligible importance can be eliminated based on sufficient evidence.

The CSM described here is in the preliminary stages of development. It includes all of the potential inputs and exposure pathways of potential receptors. At this stage, the model does not attempt to quantify the relative importance of the various processes and pathways. While a more quantitative model is desirable for evaluating remediation strategies, currently available information do not support a quantitative analysis of the relative magnitude by which each transfer mechanism contributes to exposures. The development, validation, and calibration of hydrodynamic and sediment transport models that will accurately simulate the physical and chemical processes that contribute to transport of COPCs in the river and associated environmental media will be important component of a completing the conceptual site model. Although, progress has been made in this direction (see discussion of Tidal Anacostia Model in Section 9.1), additional efforts in data collection and model development will be needed. Such models need to be developed to the point where they can support estimation of contaminant mass fluxes. This will require a more complete understanding of transport mechanisms for affected media, and, accurate estimates of contaminant loadings. The CSM described in this report model is generic with respect to constituents. As constituents differ in the degree to which they may be affected by various fate and transport processes, at some point in the risk assessment process individual chemical-specific or chemical class-specific models may need to be developed and evaluated.

The currently available data do not satisfy requirements for the development of such models. In general, data concerning chemical loadings from sources, chemical concentrations in various media, chemical transformation, and chemical transport processes have been collected over the course of several decades and throughout the Anacostia River watershed, and as such comprise a spatio-temporal patchwork picture of chemical contamination in the tidal Anacostia. In addition, analytical methods and target analytes differed between studies. The tidal Anacostia is a complex, dynamic system. Data have not been collected and analyzed in a coordinated manner in order to develop a comprehensive understanding of chemical contamination sufficient for quantifying current human exposures to various media, or for predicting future human exposures or future contamination conditions under various remediation scenarios. Some specific data gaps are itemized below.

X Some permitted and other known discharges (e.g., certain CSOs in the lower tidal Anacostia) to

Anacostia watershed water bodies are relatively well characterized. However, the Anacostia watershed, particularly the portion adjacent to the tidal Anacostia, has a long history of industrial and military activity. A detailed historical and geographic assessment of the identity, activities, and location of industrial, government, military, and research facilities in the watershed is currently unavailable, but would be useful for identifying potential locations of uncharacterized (i.e., unreported and/or unpermitted) discharges to the tidal Anacostia, its tributaries, or adjacent groundwater, and for locating additional advisable surface water and ground water sampling locations.

X NPDES permits identify allowable discharge levels of specific waste stream constituents. It is possible that other chemicals are also discharged from those facilities. Chemical loadings of contaminants that were not specified in active permits in wastewater streams from facilities holding NPDES permits are largely uncharacterized in documents available for the screening level assessment.

X Flows, first-flush and peak fluvial chemical loadings from tributaries to the tidal Anacostia during storm events have not been quantified in available documents.

X The exchange of surface water and sediment between the Potomac and Anacostia Rivers during tidal flux has not been quantified in available documents.

X The exchange of ground water and surface water within the tidal Anacostia River has not been quantified in available documents.

X Spatio-temporal human use patterns (shoreline fishing, boat fishing, boating, sailing, waterskiing, wading, swimming, etc.) at the tidal Anacostia river have not been sufficiently characterized, although consumption of contaminated fish is suspected to be a potentially significant route of human exposure to chemicals in the tidal Anacostia river (GDC, 1998 ; McCabe, 1997).

X The potential contribution of aerially deposited particles to chemical contamination in the tidal Anacostia River has not been evaluated. While locations and identity of industrial stacks with permitted emissions have been identified (McCabe, 1997), chemical composition and quantity of emitted particles and deposition patterns relative to the location of the tidal Anacostia have not been characterized in available documents.

X The frequency and extent of dredging deep tidal river sediments have not been characterized in available documents; deep dredging may promote the resuspension of formerly buried contaminated sediment.

X The Warner et al. (1997) pollution source study is the most comprehensive treatment of pollution sources to the tidal Anacostia River. The review considered total loading of several types of pollution, evaluated for only a limited number of individual chemicals, and considered loadings from only three types of sources: nonpoint stormwater runoff, CSOs, and permitted industrial and municipal discharges; however, the estimated loadings were based in whole or in part on modeling rather than entirely on site specific data.. The study did not consider loadings from ground water. This study was not sufficiently focused on chemical loadings to identify primary sources of many chemicals that are suspected to be potential problems in the river, such as PCBs, pesticides, and dioxins and dibenzofurans.

X Depositional patterns of sediments transported into the tidal Anacostia from tributaries, including the Northeast and Northwest branches, during and between storm events have not been characterized.

X Conditions under which deposited sediments are resuspended and transported, and the relative importance of this mechanism for transport of chemical contaminants, have not been characterized.

X Particulate deposition on the floodplain during storm events or during spring snowmelt in areas of the tidal Anacostia and tributaries that have not yet been channelized has not been addressed relative to human exposures to chemical contaminants.

8.2 HUMAN HEALTH SCREENING ASSESSMENT

The screening assessment utilized existing data and information to identify COPCs in the tidal Anacostia River. The overall results of the sorting of chemicals is shown in Figure 6-3. Of 215 chemicals that entered the human health screen, 43 chemicals were identified as COPCs, based on their maximum concentrations exceeding an human health RBC or ARAR. Thirty-nine are COPCs in fish tissue, 7 in river sediment and 5 in river water; six of the chemicals (or chemical mixtures) are COPCs for more than one media (Table 6-6a).

Fish tissue COPCs fall into several chemical classes. Seventeen (17) are chlorinated dibenzodioxins or dibenzofurans; 12 are organic pesticides: aldrin, (-HCH (lindane), HCB, DDT, DDE, dieldrin, chlordane (or transformation products), or heptachlor; 2 are PCBs, including Aroclor 1260; and 4 are elements: arsenic, cadmium, lead, and mercury. The remaining fish tissue COPCs, not accounted for in the latter chemical classes, are ∀-HCH, heptachlor epoxide, bis(2-ethylhexyl)phthalate, and di-N-octyl phthalate.

The 7 COPCs in sediment include two chemicals (or chemical mixtures) that are also COPCs in water and fish tissue: arsenic and total PCBs and one chemical that is also a COPC in fish tissue: Aroclor 1260. The remaining 4 sediment COPCs include the following PAHs: benzo(a)pyrene, dibenz(a,h)anthracene, benz(a)anthracene and benzo(b)fluoranthene.

The 5 COPCs in water include two chemicals (or chemical mixtures) that are also COPCs in fish tissue and sediment: total PCBs and arsenic, and three that are also COPCs in fish tissue: heptachlor (pesticide), DDE and DDT.

A high max/RBC ratio would indicate a greater potential for concern that a given chemical may pose a risk at reasonable maximal exposure (RME), given the conservative assumptions in the screening assessment (Table 6-6b). Chemicals with the highest max/RBC ratios (greater than 100) in fish tissue included total PCBs (2911), Aroclor 1260 (285), dieldrin (264) 2,3,7,8-TCDD (133) and arsenic (127). The highest max/RBC ratio in sediment was for benzo(a)pyrene (34). Total PCBs and arsenic had max/RBC ratios in surface water of 380 and 15, respectively. The three sediment COPCs that are also COPCs in fish tissue, total PCBs, Aroclor 1260, and arsenic, represent the first, second and fifth highest max/RBC in fish tissue, respectively, and all 3 had max/RBC ratios in fish tissue that exceeded 100. There was no apparent spatial trend in the distribution of either the maximum concentrations of COPCs (Figure 6-4) or frequency of detections of chemical classes represented in the COPC list (Figures 5-2 through 5-11). However, the data are not adequate for a robust analysis for spatial and temporal trends.

No chemicals could be definitively ruled out as COPCs. Of the 172 chemicals that could not be classified as COPCs, 144 were classified as Category 3 chemicals *(Insufficient Information on Exposure)* because of high uncertainty in the interpretation of the maximum concentrations (100) reported or the lack of detects (44) (Table 6-7a,b) either due to inadequate sample numbers or to inadequate geographic distribution of samples. Thirty-one chemicals were classified as Category 4 chemicals *(Insufficient Information on Toxicity)* because of the lack of an appropriate human health RBC. Additional data on exposure concentrations and toxicity will be needed in order to determine if any of these 176 chemicals are actually COPCs.

8.2.1 DATA GAPS RELATED TO EXPOSURE INFORMATION FOR THE SCREENING LEVEL RISK ASSESSMENT

The major data gaps related to exposure information used in the Anacostia River human health risk screening assessment can be classified into three categories: 1) inadequate geographic coverage of sampling in the tidal Anacostia; 2) inadequate numbers of samples; and 3) lack of data for a chemical class in a specific media.

Sediment. The database does not contain information on the concentration of contaminants in sediment upstream of Lower Beaverdam Creek. In general, there is very little information on the concentration of contaminants in sediment upstream of Benning Road Bridge and the majority of this information was collected from Kenilworth Marsh and Kingman Lake (Figures 5-2 through 5-6). More specifically, there is very little information on the concentration of metals, acid/base/neutral-extractables (ABNs) and PCBs in sediment within the Anacostia River channel, upstream of the Pennsylvania Avenue bridge.

Downstream of the Pennsylvania Avenue Bridge, the geographic coverage tends to improve with the exception of the Aroclors analyses. In the lower Anacostia, samples that were analyzed for Aroclors are limited to two areas of the river: one near and downstream of the 11th Street bridge, along the northern bank; the other area is along the southeastern bank, near the Potomac (Figure 5-4). The database does not contain information on the concentration of dioxins or furans in sediment.

Relatively few samples were analyzed for total Aroclors, ABNs and metals in areas with high detection rates for these chemical classes (i.e., Benning Road Bridge, Kenilworth Marsh and Kingman Lake) (Figures 5-9 through 5-11). These areas should be considered as potential targets for additional data collection efforts.

Approximately 73% of the sediment data contained in the database is from the analyses of samples that were collected before 1996. Since the data are not adequate to support predictions of temporal trends in sediment concentrations, additional data may need to be collected to support a human health risk screening assessment that reflects the most current conditions of the river.

Fish Tissue. There is very little information in the database on the concentration of contaminants in fish collected upstream of Watts Branch, with the exception of the pesticides and metals classes (Figure 5-4; Table 5-5). The database contains no information on the level of dioxins, furans or PAHs in fish collected north of Watts Branch.

Most of the chemical concentrations in the database were determined from the analysis of composite fish samples. Composite samples often provide a reasonable estimate of the mean concentration of a chemical but the variability of the measured concentrations will be lower. Therefore, the actual maximum concentration of a particular chemical in fish tissue may be higher than the measured concentration determined from the composite samples.

Approximately 95% of the data on chemical concentrations in fish tissue is based on samples that were collected between 1989–1995 (Cummins and Velinsky, 1993; Velinsky and Cummins, 1996).

Surface Water. Data are available for just one location upstream of the Independence Avenue Bridge (Figure 5-3). Data on the concentration of dioxins, furans and PAHs in surface water are not included in the database. There is very little information in the database on the concentration of ABNs and PCBs in surface water (Table 5-4). The data on chemical concentrations in Anacostia River water is from one study (Velinsky, 1999). The data were collected over an approximately 10 month period in 1998. The sampling events were scheduled before and after rainfall events to determine the effects of stormwater

runoff on water quality in the Anacostia River, but samples were not collected during first-flush or at peak flow.

Additional data on particulate-bound and dissolved contaminant concentrations should be collected from an array of sampling stations, at various depths and at regular time intervals to characterize spatial and temporal trends. The collection of additional data on general water quality parameters such as TSS, pH, eH, particulate and dissolved organic carbon, dissolved oxygen and temperature, should be coordinated with the collection of data on contaminant concentrations and river hydrodynamics to support the development of a hydrodynamic model of the river.

Background. The database does not contain information on background concentrations. For purposes of a screening level human health risk assessment, U.S. EPA risk assessment guidance suggests inorganic chemicals present at naturally occurring background levels can be removed from the COPC list (U.S. EPA, 1989). Note that the U.S. EPA guidance (U.S. EPA, 1989) specifies that comparing concentrations of organic chemicals detected at a site to naturally occurring concentrations or anthropogenic levels (non-site related concentrations) is inappropriate at the screening level risk assessment.

Estimation of Concentration Term Parameters for Risk Estimates. An additional issue for use of the existing data in a human health risk assessment is the lack of adequate information on detection and sample quantitation limits for the various analytes captured in the database. This limitation has no effect on the human health screening assessment, which is based entirely on maximum concentrations detected; however, it may severely compromise the estimation of statistical parameters such as the mean and associated confidence limits. The latter would be needed to estimate the concentration terms for each chemical that would enter the calculations of baseline risk.

8.2.2 DATA GAPS RELATED TO TOXICITY INFORMATION

Thirty-one chemicals could not be evaluated against toxicity criteria because of the lack of an RBC (Table 6-8). Had RBCs been available for these chemicals, it is possible that some would have had maximum concentrations that exceeded their respective RBCs. Many of the chemicals in this category are structural analogs, similar mixtures or are toxicologically similar to other COPCs. For example, 12 chemicals in this category are PAHs which, as a chemical class, are represented on the COPC list. Twelve chemicals are pesticides or structural analogs, including several structural or compositional analogs of chemicals that are COPCs: BHCs, hexachlorocyclohexane-delta, the ortho-para isomers of DDD, DDE and DDT, oxychlordane. At least one chemical, arsenic III, can probably be eliminated from this list based on an interpretation of the RBC for arsenic to apply to all forms of inorganic arsenic. It may also be possible to eliminate several of the PAHs and pesticides (e.g., the various lindane-like mixtures and the ortho-para isomers of DDD, DDE and DDT) from this category based on their toxicologic similarity (or an assumed similarity as a conservative assumption) to other chemicals for which RBCs are available. It is also possible that a search of existing toxicology literature on these chemicals may discover data or reference values that would be adequate to support a provisional RBC for use in the screening assessment. Potential sources for reference values may be the U.S. EPA Superfund Health Risk Technical Support Center; the U.S. EPA Health Effects Assessment Summary Tables (HEAST), ATSDR or WHO.

8.3 ECOLOGICAL SCREENING ASSESSMENT

8.3.1 BENTHIC INVERTEBRATES ASSESSMENT

The potential for effects in benthic invertebrates was assessed by comparing the maximum and mean concentrations of COPCs in sediment to corresponding screening benchmarks (TELs and PELs). The results of this screening were HQs calculated as the ratio of the sediment concentration to the corresponding screening benchmark.

In the Upper River, TEL-HQ values greater than 100 were calculated for the maximum and mean sediment barium concentrations. TEL-HQs greater than 20 were calculated for maximum concentrations of all the PAH compounds and several pesticides, chlordane, DDD, DDE, and DDT. The only PEL-HQ greater than 20 was calculated for chlordane.

In the Lower River, TEL-HQ values greater than 100 were calculated for the maximum and mean concentrations of barium, many of the PAH compounds, and maximum Aroclor 1260 and total PCB concentrations. PEL-HQs greater than 100 were calculated for the maximum concentrations of three PAH compounds, benzo(a)anthracene, pyrene, and phenanthrene.

Finally, in the Ship Channel, TEL-HQ values greater than 100 were calculated for the maximum concentrations of lead, all of the PAH compounds, total PCBs, DDE, and total DDTs. PEL-HQs greater than 20 were calculated for maximum concentrations of lead, benzo(a)anthracene, phenanthrene, LPAHs, HPAHs, total PAHs, DDD and DDE.

Limited toxicity testing has been conducted with sediments collected from both Kenilworth Marsh and the Anacostia River. Significant toxicity was observed with a subset of the samples collected from both locations.

The results of this assessment suggest that exposure to COPCs in Anacostia River sediments may result in deleterious effects in benthic invertebrates.

8.3.2 FISH ASSESSMENT

The exposure of fish in the Anacostia River to COPCs was evaluated using three approaches. First, surface water contaminant concentrations were compared to AWQC. Then, tissue concentrations were compared to tissue concentrations associated with adverse impacts in freshwater fish. Finally, the exposure to PAHs was evaluated by comparing the measured sediment PAH concentrations to sediment concentrations associated with adverse effects.

In the Upper River, the maximum dissolved lead and total PCB concentrations exceeded the corresponding AWQC with HQs less than 2.

The maximum measured fish tissue concentrations in the brown bullhead and the largemouth bass exceeded the corresponding LOEC value in the Upper River, the Lower River and the Ship Channel. In addition, maximum lead tissue concentrations exceeded the corresponding LOEC in both the Upper River and the Lower River. Finally, tissue concentrations of three pesticides, dieldrin, DDD, and DDE exceeded the corresponding LOEC values in the Lower River and the Ship Channel.

Maximum sediment concentrations in all three areas exceeded the PAH sediment screening threshold value of 2 :g/g. The highest sediment PAH concentrations were measured in the lower river zone. Mean sediment total PAH concentrations in all three areas also exceeded the threshold value.

The results of this assessment suggest that fish tissue concentrations of PCBs, lead, and pesticides may indicate the potential for adverse effects due to exposure to these contaminants. Finally, sediment PAH concentrations throughout the lower Anacostia River are higher than concentrations associated with adverse effects in benthic fish.

8.3.3 AQUATIC BIRD ASSESSMENT

The exposure of aquatic birds to COPCs associated with the Anacostia River was estimated based on the assumption that the contaminant exposure was entirely through dietary exposure. The risk associated with dioxin and furan exposure to the green heron was estimated using a TEQ based approach. The estimated doses of the other COPCs to the green heron were compared to TRVs derived for the great blue heron (Sample et al. 1996).

The doses of dioxins and furans, when calculated as TEQs, for the Upper River and Lower River were higher than the corresponding NOAEL TEQ values for the green heron. However, these doses did not exceed LOAEL TEQ values.

The estimated doses of methyl mercury and total DDT to green heron in all three areas were greater than the TRV values for doses calculated either on maximum or mean fish tissue concentrations. In addition, the calculated dose of lead to green heron in the lower river based on the maximum lead concentrations exceeded the corresponding TRV. Finally, the dose of total PCBs calculated using both maximum and mean fish tissue concentrations in the Ship Channel exceeded TRV values for the green heron.

The results of this assessment suggest that dioxins and furans, lead, methyl mercury, total DDT, and PCBs are present in fish tissues within the lower Anacostia River at concentrations that may result in adverse impacts on the green heron. Some of these risk calculations are likely biased high however. For instance, methyl mercury concentrations were not available so total mercury was used as a surrogate. The diet of the great blue heron, which has a greater aquatic component than the green heron, was applied using the smaller body weight of the green heron. Also, complete assimilation of ingested COPCs was assumed.

8.3.4 AQUATIC MAMMAL ASSESSMENT

The exposure of a mammal, the raccoon, to COPCs associated with the Anacostia River was estimated based on the assumption that the contaminant exposure was entirely through dietary exposure. The risk associated with dioxin and furan exposure to the raccoon was estimated using a TEQ based approach. The estimated doses of the other COPCs to the raccoon were compared to TRVs derived for the mink (Sample et al. 1996).

Calculated doses of total PCBs and certain trace elements for the raccoon in all three areas were greater than TRV values when the maximum tissue and sediment concentrations were used to calculate the dose. The highest HQ values were calculated for trace elements in the lower river zone. When average tissue and sediment concentrations were used to calculate the dose for the raccoon, TRV values were exceeded for certain trace elements in all three areas. The TRV for total PCBs were also exceeded using average values for the Washington Ship Channel/Tidal Basin zone. The PCB TRV was not exceeded by estimated doses based upon averages from either the upper or lower river zone.

A large range in the diet of raccoons can be found in the literature. For initial, conservative screening, a diet that emphasizes aquatic prey, especially fish, was used for estimating COPC doses. An alternative diet reported for raccoons in Maryland, for which fish comprise only 3% of the total diet, was also used to calculate alternative COPC doses. Using this alternative diet with maximum exposure estimates, TRVs for certain metals were exceeded in all three zones and the PCB TRV was exceeded only by estimated

doses for the lower river zone.

The results of this assessment suggest that trace elements and PCBs associated with sediments and fish tissues may possibly be high enough within the lower Anacostia River to result in adverse effects for the raccoon. There is a fair degree of variability and uncertainty associated with this indication however. Actual use of the habitat, the specific diet of animals in the Anacostia, plus the bioavailability and assimilation efficiency of ingested COPCs are major factors which would influence the actual risk to aquatic wildlife as represented by the raccoon.

8.4 POTENTIAL SOURCES OF COPCS

As discussed in Section 2.2.5, current point sources of ongoing chemical release to the tidal river may include releases from current activity (e.g., pesticide application) as well as transport of chemicals from point sources of previous contamination (e.g., PCB migration in runoff from contaminated soil). Both above-ground and subsurface releases may occur; subsurface releases may result from, for example, leaching of BTEX from a leaking underground storage tank to groundwater followed by seepage into the river. From the information currently available, it is not possible to assign specific COPCs or chemical classes to a specific source or activity. It is also not possible from the data available, to discern point sources of a particular contaminant that also has significant non-point sources because insufficient information is available to determine if a pattern is present. However, it is possible to assign COPC chemical classes to types of activities and processes that are known to have occurred in the watershed. Whether these activities and processes actually have contributed COPCs to the tidal Anacostia River, or the extent of any contribution cannot be ascertained from the information evaluated in this assessment.

Potential local, current and/or historical, sources of release of mercury include production and disposal of alkaline batteries, electrical switches, certain types of lighting discharge tubes, and various medical devices, including thermometers, manometers and amalgam dental fillings materials. Mercury is also used in the manufacture of plastics, including vinyl chloride polymers, chlorine, and caustic soda. Historically, mercury compounds have been used as paint pigments and fungicides, and in the extraction and purification of gold Mercury is released to the air during the combustion of fossil fuels (ATSDR, 19##).

Potential local, current and/or historical, sources of release of PCBs include power utilities, which historically have used PCBs in electrical capacitors and transformers, and any other activities involving the production, installation, maintenance or removal of capacitors and transformers. The latter would include local facility power plants, including those on ships. PCBs have also been used as plasticisers, surface coatings, and inks, as pesticide extenders and in carbonless duplicating paper (ATSDR 19##).

Potential local, current and/or historical, sources of release of PAHs include incomplete combustion of fossil fuels, including automobiles, wood stoves and furnaces, and fossil fuel power plants; and transport, storage or processing of crude oil, shale oil, coal tar and other petroleum containing materials. Natural wood fires can also release PAHs to the air (ATSDR, 19##).

Potential local, current and/or historical, sources of release of CDDs include incineration of chlorine-containing materials (including plastics, wood and paper) and handling and disposal of ash from such facilities. Natural wood fires can also release CDDs to the air. CDDs can also be produced a side-product in the manufacture of chlorinated phenols (ATSDR, 19##).

Potential local, current and/or historical, sources of release of lead include copper, silver and lead smelting; combustion of leaded gasoline; leaching and transport of dust and debris from surfaces coated with lead-based paints, including older houses, bridges, ships; leachate and transport from firing ranges; and manufacture, disposal, storage or reprocessing of lead-acid batteries, electrical conduit and other

products containing lead. (ATSDR, 19##).

Potential local, current and/or historical, sources of release of arsenic may include copper and lead smelting operations, arsenic-containing pesticides and wood preservatives, and leachate from older cemeteries (arsenic-containing materials were historically used as a tissue preservative). (ATSDR 19##).

Potential sources of the various pesticides that were identified as COPCs would include commercial or residential agriculture and lawn care, and facility pest control processes.

REFERENCES FOR CHAPTER 8

ATSDR. 1998. Toxicological profile for polychlorinated biphenyls (PCBs). Atlanta, GA: U.S. Department of Health & Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR. 1989. Toxicological profile for 2,3,7,8-tetra dibenzo-p-dioxins (TCDDs). Atlanta, GA: U.S. Department of Health & Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR. 1990. Toxicological profile for polycyclic aromatic hydrocarbons (PAHs). Atlanta, GA: U.S. Department of Health & Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR. 2000. Toxicological profile for arsenic. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

ATSDR. 1999. Toxicological profile for lead. Agency for Toxic Substances and Disease Registry. Atlanta, GA.

ATSDR. 1999. Toxicological Profile for Mercury. Atlanta, GA: Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services.

9. RECOMMENDATION FOR FUTURE ACTION

Data gaps identified in Section 8 are considered here in terms of possible targets for future activities in Phase II of a risk characterization of the tidal Anacostia River. Specific requirements for data collection are not provided, and would be developed in the scoping of Phase II activities.

9.1 CONCEPTUAL SITE MODEL AND PREDICTIVE MODEL OF SITE CONTAMINATION AND HUMAN HEALTH RISKS

The ultimate goal of the risk assessment process is to achieve a sufficiently detailed understanding of the tidal Anacostia River so that 1) current ecological and human health risks can be reliably estimated; and 2) changes in risk that may attend remediation strategies can be predicted with sufficient confidence to support risk management decisions. The latter goal demands the development, validation, and calibration of hydrodynamic and a sediment transport models that will accurately simulate the physical and chemical processes that contribute to transport of COPCs in the river and associated environmental media. Such models need to be developed to the point where they can support estimation of contaminant mass fluxes. This will require a more complete understanding of transport mechanisms for affected media, and accurate estimates of contaminant loadings.

At this time, the available data and information fall short of what is needed to achieve these objectives, in part because they do not adequately address the dynamic nature of the tidal Anacostia River. Major data gaps have been summarized in Section 8 and elsewhere in this report and will not be reiterated in detail here; however, the important limitations of the available data, in terms of estimating and predicting risks, are as follows:

X Spatial and temporal profiles of the chemical concentrations in the sediment and water column have not been sufficiently characterized to identify chemicals of concern or to estimate exposure concentrations of these chemicals.

X Chemical inputs from major point sources (including aerial sources) and from non-point sources (including ground water) have not been adequately identified, characterized and quantified to support quantitative models of loadings to the river.

X The spatial distribution, magnitude and mobility of historical contamination of sediments and ground water are not sufficiently understood to model their contribution to contamination of surficial sediments or the water column.

X The hydrodynamics and sediment transport in the river are not understood sufficiently to enable predictions of contaminant concentration profiles over the length of the river, or to identify future high impact areas and estimate associated concentrations. This limitation is particularly relevant to extreme (high and low) flow conditions and disturbances that might be imposed on the river as part of remediation or other modifications to the river (e.g., dredging).

X Spatial and temporal patterns of human uses of the river, including shoreline fishing, boat fishing,

boating, sailing, waterskiing, wading, swimming, picnicking (etc.) at the tidal Anacostia river have not been sufficiently characterized to ensure that all potential human receptors are considered in risk estimates, or to estimate values for exposure factors for potential exposure scenarios.

Based on the above considerations, a long-term, multi-year sampling program is recommended for satisfying the data needs for risk characterization as well as predictive modeling of the river. Such efforts could be considered as targets for data collection in a second phase of this risk assessment. Ideally, this program should have the following major features:

X Sediment and water sampling should include target areas of the river immediately downstream from major inflows and suspected point sources or inputs from non-point sources (e.g., sewer pipes and flood channels), as well as those areas of the tidal river where particulate deposition is demonstrably greatest.

X The sediment sampling design should use a stratified random or adaptive sampling approach to support unbiased parameter estimates of exposure concentrations (e.g., mean and confidence limits). The design should ensure collection of adequate numbers of samples and eliminate or account for sampling bias (temporal, spatial, nonrandomness) that might affect the parameter estimates.

X Surface water samples should be collected during extreme (high and low) flow conditions and during disturbances of the sediment by other anticipated human activities (e.g. dredging).

X Water flows and channel volumes should be determined over a sufficient geographic and temporal scale to support the development of a hydrodynamic model of both the “average” long-term behavior of the river and the behavior of the river during extreme events (e.g., storms, drought).

X A model calibration data set should be collected. This would include sediment and water column concentrations of representative chemicals at various locations in the river, including predicted high impact areas, at various times, including during and after extreme events (e.g. storms).

X Surveys of human uses of the tidal river area should be conducted throughout at least one annual cycle.

Future sampling efforts in the Anacostia watershed by AWTA members and other organizations should be coordinated to avoid duplication of effort.

The above sampling program would provide data to satisfy both the requirements for estimating current human health risks and the requirements for model development and calibration. The development of a preliminary model early in the sampling program would be highly desirable, as the model may identify other important data collection needs. Progress towards developing hydrodynamic and sediment transport models of the tidal Anacostia River may benefit from earlier and on-going efforts. For example, a one-dimensional hydrodynamic model was developed in 1988 as a component of the Tidal Anacostia Model (TAM), which has been used in several past efforts to study dissolved oxygen levels in the river. This includes the District of Columbia's Total Maximum Daily Load (TMDL) program which models for dissolved oxygen, fecal coliforms, and suspended solids and which incorporates the hydrodynamic component of TAM, and modified versions of the U.S. EPA's Water Quality Analysis Simulation

Program (WASP). The TAM/WASP model for suspended solids also incorporates algorithms for simulating the deposition and re-suspension of sediments from the U.S. EPA's HSPF (Hydrologic Simulation Program FORTRAN) model. The major inputs to the TAM hydrodynamic model include estimates of daily flows from the NE and NW Branches, CSO's, tributaries, and storm sewers, hourly tidal heights at the downstream boundary of the model (confluence with the Potomac), and model segment geometry, including tidally-averaged depths and cross-sectional areas of the sixteen model transects.

9.2 SPECIFIC IMPROVEMENTS AND ENHANCEMENTS TO DATA COLLECTION EFFORTS

In addition to the above more general recommendations, specific recommendations regarding data collection approaches that would greatly enhance the value of any additional data that is collected, in terms of its use in human health risk screening or risk estimation, include the following:

Compositing of samples and sampling design.

X Some of the sediment and fish tissue chemical concentrations in the database were determined from the analysis of grab samples while others were determined from composite samples. While composite samples are useful for determining average concentrations of chemicals, some information on the variability of the chemical concentration is lost. With respect to sediment samples, information on concentration variability for individual grab samples is useful for identifying areas of high contamination (i.e., hot spots), mapping chemical concentrations and predicting concentrations in unsampled locations. Accurate estimates of the variability of chemical concentrations are particularly important if the concentration data will be used to develop hydrodynamic models of the watershed. Underestimating the variability of measured or predicted chemical concentrations will tend to overestimate the precision in model predictions, thereby underestimating the uncertainty in decisions that are based on those model predictions.

X An issue related to sample compositing is the relationship between sample size and the variance of measured concentrations. The sampling plan design to support risk estimates should consider the interactions between sample volume and the variability of the measured concentrations. As the sample volume increases, the variance of the measured concentrations will tend to decrease. The relationship between sample volume and variance effects not only the reasonable maximum exposure concentration used in risk assessment but also decisions made during the remedial investigation and remedial design phases. For this reason it is important that the sampling plan design specify sample sizes that are appropriate for the data quality objectives (DQOs) developed for a site.

Sediment. As noted in Section 8, approximately 73% of the data on the concentration of chemicals in sediment was collected prior to 1996. Additional sediment samples should be collected throughout the Anacostia River to update the information contained in the database. Detailed suggestions for additional sediment sampling efforts are as follows:

X Collect additional sediment samples in the upper Anacostia (north of the Pennsylvania Avenue Bridge); analyze for the seven chemical classes: ABNs, dioxins, furans, metals, PAHs, total PCBs, PCB Aroclors and pesticides.

X Characterize the spatial distribution of total PCBs and PCB Aroclors in the lower as well as the upper Anacostia.

X Characterize the spatial distribution of the concentration of dioxins and furans in Anacostia River sediment.

X Collect additional samples in areas with apparently high detection rates that were sparsely sampled: Kenilworth Marsh (ABNs, total PCBs, PCB Aroclors) and the Anacostia River channel north of the Pennsylvania Ave. Bridge (ABNs, metals).

X Characterize the bioavailability and risk of sediment contamination through toxicity testing concurrent with chemical analysis at sufficient spatial scale to characterize areas of elevated risk.

X Identify major sediment deposits and probe for depth estimates. Collect core samples to evaluate sediment types and sediment coring techniques required for sampling large-scale sampling.

X Analyze of sediment transport to identify potential depositional, scour, and source areas in order to provide a sound basis for interim and final remedial decisions, including possible recontamination issues.

Fish tissue. Approximately 95% of the data on the concentration of chemicals in fish tissue was collected between 1989–1995. Additional fish tissue samples should be collected throughout the Anacostia River to update the information contained in the database. Based on the data gaps identified in Section 8, the following recommendations for additional fish tissue sampling efforts are provided:

X Future sampling efforts should ensure that an adequate quantity of fish tissue samples are collected north of Watts Branch; additional data are needed on the concentration of chemicals in fish tissue samples for all the chemical classes previously defined.

X Individual samples should be collected and analyzed so the variability of chemical concentrations in fish tissue can be characterized.

X The coordinates of the fish sampling locations should be provided so spatial analyses of the data can be performed as part of the risk assessment.

X In order to refine the relationships between tissue residue levels with both observed impacts and sediment contamination, more detailed information on the migratory habits of selected species should be developed.

Surface water. The data on chemical concentrations in Anacostia River water were collected over an approximately 10-month period in 1998. Additional data should be collected to determine the spatial and temporal distribution of chemicals in the Anacostia River. Based on the data gaps identified in Section 8, additional recommendations are:

X The concentration of dioxins, furans and PAHs in surface water should be determined.

X Additional data should be collected to determine the concentration of chemicals at surface water sampling stations located north of the Independence Ave Bridge.

X Additional data should be collected to determine the concentration of ABNs and PCBs in surface water throughout the tidal Anacostia River.

X Additional data on particulate-bound and dissolved contaminant concentrations should be collected from an array of sampling stations, at various depths and at regular time intervals to characterize spatial and temporal trends. The collection of additional data on general water quality parameters such as TSS, pH, eH (redox potential), particulate and dissolved organic carbon, dissolved oxygen and temperature, should be coordinated with the collection of data on contaminant concentrations and river hydrodynamics to support the development of a hydrodynamic model of the river.

Background chemical concentrations. Data on naturally occurring and local background concentrations of inorganic chemicals in each media (i.e., surface water, sediment, fish tissue) should be collected..

Chemical loadings. The following specific types of information are needed:

X Flow rates from tributaries and point discharges, including CSOs, should be quantified.

X First-flush and peak fluvial chemical loadings from tributaries to the tidal Anacostia during storm events should be quantified.

X A detailed historical and geographic assessment of the identity, activities, and location of industrial, government, military, and research facilities in the watershed would be useful for identifying potential locations of uncharacterized (i.e., unreported and/or unpermitted) discharges to the tidal Anacostia, its tributaries, or adjacent groundwater, and for locating additional advisable surface water and ground water sampling locations.

X Chemical composition, quantity of emissions, and particulate deposition patterns from known aerial point sources should be determined in order to assess the potential contribution of aerially deposited particles to chemical contamination in the tidal Anacostia River.

X Potential loadings from ground water should be quantified.

X A detailed historical and geographic assessment of the identity, activities, and location of industrial, government, military, and research facilities in the watershed would be useful for identifying potential locations of uncharacterized (i.e., unreported and/or unpermitted) discharges to the tidal Anacostia, its tributaries, or adjacent groundwater, and for locating additional advisable surface water and ground water sampling locations.

X A detailed historical review of the frequency and extent of dredging deep tidal river sediments would be useful for identifying impacted areas and targeting sampling.

Hydrodynamics and sediment transport. The following information is needed to support predictive modeling of exposure concentrations:

X Rates and amounts of exchange of surface water and sediment between the Potomac and Anacostia Rivers during tidal flux.

X Rates and amounts of exchange of ground water and surface water within the tidal Anacostia

River.

X Water flows and channel volumes should be determined over a sufficient geographic and temporal scale to support the development of a hydrodynamic model of both the "average" long-term behavior of the river and the behavior of the river during extreme events (e.g., storms, drought).

X Depositional patterns of sediments transported into the tidal Anacostia from tributaries, including the Northeast and Northwest branches, during and between storm events.

X Deposition of sediments on the floodplain, particularly within Kenilworth Marsh and Kingman Lake, during large flow events

X Conditions under which deposited sediments are resuspended and transported, and the relative importance of this mechanism for transport of chemical contaminants.

X Particulate deposition on the floodplain during storm events or during spring snow melt in areas of the tidal Anacostia and tributaries that have not yet been channeled.

X Partitioning of COPCs between sediment and surface water

X Degradation rates or environmental half-times of COPCs

Potential receptors and behavior. The following specific types of information are needed to characterize receptors and exposures to receptors:

X Spatial and temporal patterns of human use of the river channel and shoreline are needed. For example, estimates of site-specific estimates of seasonal consumption of locally harvested fish are needed (amounts and species) are needed. Estimates of the frequency and types of shoreline uses of the river are needed.

X Bioavailability and bioaccumulation potential to environmental receptors should be characterized in such a manner as to identify source areas. This might be accomplished through in situ deployment of caged organisms or semi-permeable membrane devices. This would complement loading information by indicating whether and where contaminants released are bioavailable, plus provide information on the relative importance of different types of sources.

X Larval anadromous fish originating from spawning and nursery grounds throughout the Anacostia watershed may be at risk to exposure to contamination during outmigration. The success of planned restocking/restoration efforts for certain anadromous species may be compromised if the viability of outmigrating larvae is severely diminished. This potential risk might be estimated by either in situ or laboratory toxicity tests. In situ testing could also be designed to integrate cumulative, individual exposure episodes and thus provide a more complete estimation of risk.

9.3 IMPROVEMENTS TO THE EXISTING DATABASE.

If the existing data are to be used to estimate human health risks, a thorough review of the quality of the historic analytical data are needed. This review should include the verification and reporting of detection and/or sample quantification limits on all analytical results so that appropriate surrogate values can be assigned to reported non-detects. This will be needed to estimate parameters of the exposure concentrations (e.g., mean and confidence limits).

APPENDIX A - TABLES

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
83329	Acenaphthene	45	39 / 45	8.53E-03	2.20E+01	1.79E+00	4.71E+00
208968	Acenaphthylene	45	34 / 45	4.23E-03	2.90E+00	4.76E-01	7.24E-01
67641	Acetone	22	11 / 22	1.20E-02	2.90E-01	8.00E-02	8.25E-02
309002	Aldrin	42	15 / 42	4.00E-05	4.90E-02	9.03E-03	1.11E-02
7429905	Aluminum	34	33 / 34	1.01E+03	9.47E+04	1.17E+04	1.57E+04
120127	Anthracene	45	36 / 45	1.97E-02	5.80E+00	6.87E-01	1.26E+00
7440360	Antimony	23	3 / 23	4.80E-01	5.50E+00	2.14E+00	1.76E+00
12674112	Aroclor 1016	14		4.40E-02	4.90E-01	1.69E-01	1.33E-01
11104282	Aroclor 1221	14		8.90E-02	4.90E-01	2.10E-01	1.11E-01
11141165	Aroclor 1232	14		4.40E-02	4.90E-01	1.69E-01	1.33E-01
53469219	Aroclor 1242	16		4.40E-02	4.90E-01	1.54E-01	1.30E-01
12672296	Aroclor 1248	16		4.40E-02	4.90E-01	1.54E-01	1.30E-01
11097691	Aroclor 1254	25	11 / 25	4.40E-02	1.63E+00	4.42E-01	4.16E-01
11096825	Aroclor 1260	25	15 / 25	1.80E-03	1.20E+01	6.64E-01	2.38E+00
7440382	Arsenic	34	33 / 34	1.30E+00	2.69E+01	6.23E+00	4.30E+00
111444	Bis(2chloroethyl)ether	22		3.80E-01	2.30E+00	7.02E-01	4.10E-01
39638329	Bis(2chloroisopropyl) ether	8		4.50E-01	6.60E-01	5.09E-01	8.03E-02
117817	Bis(2ethylhexyl) phthalate	22	20 / 22	7.10E-02	7.20E+00	2.02E+00	2.04E+00
56553	Benz(a)anthracene	45	44 / 45	6.80E-02	1.60E+01	1.85E+00	3.17E+00
53703	Dibenz(a,h)anthracene	45	37 / 45	2.80E-02	6.90E+00	6.12E-01	1.20E+00
50328	Benzo(a)pyrene	45	44 / 45	6.90E-02	2.70E+01	2.52E+00	5.17E+00
7440393	Barium	33	33 / 33	3.19E+01	1.70E+02	1.12E+02	3.45E+01
205992	Benzo(b)fluoranthene	44	41 / 44	1.20E-01	9.20E+00	1.60E+00	2.07E+00
	Benzo(b,k)fluoranthene	1	1 / 1	5.37E-01	5.37E-01	5.37E-01	
111911	Bis(2chloroethoxy)methane	22		3.80E-01	2.30E+00	6.76E-01	4.20E-01
71432	Benzene	22	1 / 22	6.00E-04	1.50E-02	7.25E-03	5.43E-03
65850	Benzoic acid	13		2.00E+00	2.20E+01	1.14E+01	7.49E+00
100516	Benzyl alcohol	20		4.10E-01	4.30E+00	1.85E+00	1.35E+00
192972	Benzo(e)pyrene	23	23 / 23	1.26E-01	7.63E-01	4.28E-01	1.73E-01

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
7440417	Beryllium	33	30 / 33	3.10E-01	3.07E+00	1.31E+00	6.88E-01
191242	Benzo(g,h,i)perylene	45	39 / 45	8.00E-02	1.30E+01	1.28E+00	2.33E+00
92524	Biphenyl	23	15 / 23	1.41E-03	4.69E-02	1.59E-02	8.89E-03
207089	Benzo(k)fluoranthene	45	41 / 45	9.30E-02	9.50E+00	1.38E+00	1.98E+00
7440428	Boron	11	11 / 11	1.60E+01	3.58E+01	2.27E+01	5.30E+00
101553	4Bromophenyl phenyl ether	22		3.80E-01	2.30E+00	9.71E-01	4.81E-01
124481	Dibromochloromethane	22		2.00E-04	1.50E-02	6.91E-03	5.77E-03
74839	Bromomethane	22		6.00E-04	3.00E-02	1.27E-02	1.10E-02
75252	Bromoform	22		1.00E-04	1.50E-02	6.86E-03	5.83E-03
78933	2Butanone	22	6 / 22	1.60E-03	7.20E-02	2.35E-02	1.69E-02
85687	Butylbenzyl phthalate	22	3 / 22	1.00E-01	2.30E+00	6.77E-01	4.47E-01
156592	cis1,2Dichlorethene	8		4.00E-04	6.00E-04	4.63E-04	9.16E-05
10061015	cis1,3Dichloropropene	22		4.00E-04	1.50E-02	6.98E-03	5.69E-03
7440439	Cadmium	54	47 / 54	2.80E-01	3.18E+00	1.60E+00	6.16E-01
7440702	Calcium	22	22 / 22	2.09E+03	2.18E+05	1.61E+04	4.56E+04
86748	Carbazole	2		3.80E-01	4.00E-01	3.90E-01	1.41E-02
56235	Carbon tetrachloride	22		5.00E-04	1.50E-02	7.02E-03	5.64E-03
5103742	Transchlordane	14	14 / 14	7.16E-03	8.05E-02	2.88E-02	2.29E-02
27304138	Oxychlordane	30	14 / 30	7.30E-04	1.74E-02	6.07E-03	5.30E-03
5103719	Chlordane alpha	45	31 / 45	2.02E-03	4.90E-01	6.42E-02	1.00E-01
5566347	Chlordane gamma	44	30 / 44	2.30E-03	4.90E-01	7.03E-02	9.98E-02
67663	Chloroform	22		5.00E-04	1.50E-02	7.03E-03	5.63E-03
2921882	Chlorpyrifos	2	2 / 2	1.06E-03	2.76E-03	1.91E-03	1.20E-03
	Chromium, total	47	47 / 47	6.90E-02	1.23E+02	3.44E+01	2.96E+01
218019	Chrysene	45	44 / 45	8.10E-02	1.60E+01	2.17E+00	3.34E+00
319846	Hexachlorocyclohexanealpha	44	6 / 44	5.00E-05	4.90E-02	8.69E-03	1.18E-02
319857	Hexachlorocyclohexanebeta	44	7 / 44	1.00E-04	4.90E-02	8.54E-03	1.18E-02
319868	Hexachlorocyclohexanedelta	44	5 / 44	5.00E-05	4.90E-02	9.02E-03	1.22E-02
58899	Hexachlorocyclohexanegamma (Lindane)	44	8 / 44	9.00E-05	4.90E-02	7.98E-03	1.18E-02

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
59507	4Chloro3methylphenol	22		3.80E-01	2.30E+00	7.72E-01	3.98E-01
106478	4Chloroaniline	22		3.80E-01	4.30E+00	1.72E+00	1.36E+00
108907	Chlorobenzene	22	3 / 22	3.00E-04	2.00E-02	8.01E-03	5.96E-03
95501	1,2Dichlorobenzene	22		3.80E-01	2.40E+00	1.17E+00	6.73E-01
120821	1,2,4Trichlorobenzene	22	1 / 22	3.80E-01	2.30E+00	8.25E-01	4.08E-01
541731	1,3Dichlorobenzene	22		3.80E-01	2.30E+00	1.02E+00	5.17E-01
106467	1,4Dichlorobenzene	22		3.80E-01	2.30E+00	1.01E+00	5.13E-01
608935	Pentachlorobenzene	6	5 / 6	1.20E-04	7.50E-04	4.73E-04	2.19E-04
118741	Hexachlorobenzene (HCB)	52	10 / 52	4.00E-05	2.30E+00	3.79E-01	4.68E-01
75274	Bromodichloromethane	22		4.00E-04	1.50E-02	7.01E-03	5.65E-03
87683	Hexachlorobutadiene	22		3.80E-01	2.30E+00	9.16E-01	4.46E-01
	1,2,4,4 Tetrachlorobenzene	6	6 / 6	3.60E-04	1.79E-03	1.04E-03	4.61E-04
95943	1,2,4,5Tetrachlorobenzene	6	6 / 6	3.80E-04	3.11E-03	1.97E-03	1.07E-03
91941	3,3'Dichlorobenzidine	22		3.80E-01	8.70E+00	3.07E+00	2.92E+00
77474	Hexachlorocyclopentadiene	22		3.80E-01	2.30E+00	9.16E-01	4.46E-01
79345	1,1,2,2Tetrachloroethane	22		3.00E-04	1.50E-02	6.96E-03	5.71E-03
75003	Chloroethane	22		8.00E-04	3.00E-02	1.28E-02	1.09E-02
75343	1,1Dichloroethane	22		5.00E-04	1.50E-02	7.02E-03	5.64E-03
71556	1,1,1Trichloroethane	22		5.00E-04	1.50E-02	7.02E-03	5.64E-03
79005	1,1,2Trichloroethane	22		4.00E-04	1.50E-02	6.98E-03	5.69E-03
107062	1,2Dichloroethane	22		3.00E-04	1.50E-02	6.97E-03	5.70E-03
67721	Hexachloroethane	22		3.80E-01	2.30E+00	9.48E-01	4.66E-01
75354	1,1Dichloroethene	22		1.00E-03	1.50E-02	7.22E-03	5.41E-03
540590	1,2Dichloroethene	14		6.00E-03	1.50E-02	1.07E-02	3.31E-03
79016	Trichloroethene	22		1.20E-03	1.50E-02	7.33E-03	5.28E-03
127184	Tetrachloroethylene	8		6.00E-04	8.00E-04	6.50E-04	9.26E-05
110758	2Chloroethylvinyl ether	8		6.00E-04	9.00E-04	6.75E-04	1.16E-04
74873	Chloromethane	22		1.10E-03	3.00E-02	1.30E-02	1.07E-02
91587	2Chloronaphthalene	22		3.80E-01	2.30E+00	8.14E-01	4.00E-01
95578	2Chlorophenol	22		3.80E-01	2.30E+00	9.85E-01	4.91E-01

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
120832	2,4Dichlorophenol	22		3.80E-01	2.30E+00	8.66E-01	4.17E-01
95954	2,4,5Trichlorophenol	22		9.30E-01	5.90E+00	1.94E+00	1.18E+00
88062	2,4,6Trichlorophenol	22		3.80E-01	2.30E+00	8.95E-01	4.31E-01
87865	Pentachlorophenol	22		9.30E-01	5.90E+00	2.30E+00	1.00E+00
78875	1,2Dichloropropane	22		5.00E-04	1.50E-02	7.05E-03	5.61E-03
7440484	Cobalt	22	21 / 22	2.60E+00	2.26E+01	1.31E+01	5.86E+00
7440508	Copper	47	47 / 47	1.62E+01	6.31E+02	8.97E+01	9.70E+01
7005723	4Chlorophenyl phenyl ether	22		3.40E-01	2.30E+00	6.36E-01	4.40E-01
75150	Carbon disulfide	22		8.00E-04	1.50E-02	7.13E-03	5.51E-03
57125	Cyanide	22	3 / 22	5.80E-01	4.88E+01	5.65E+00	1.22E+01
84662	Diethyl phthalate	22		3.80E-01	2.30E+00	6.94E-01	4.12E-01
132649	Dibenzofuran	23	14 / 23	4.37E-02	4.30E+00	1.05E+00	1.20E+00
132650	Dibenzothiophene	22	21 / 22	9.69E-03	1.04E-01	3.52E-02	2.07E-02
1002535	Dibutyl tin	1	1 / 1	5.00E-03	5.00E-03	5.00E-03	
115322	Dicofol	6	6 / 6	1.60E-04	4.28E-03	1.71E-03	1.43E-03
60571	Dieldrin	45	25 / 45	4.00E-05	9.90E-02	1.10E-02	2.08E-02
84742	Dinbutyl phthalate	22	3 / 22	1.30E-01	2.30E+00	9.20E-01	5.05E-01
131113	Dimethyl phthalate	22	1 / 22	8.20E-02	2.30E+00	6.31E-01	4.49E-01
534521	4,6dinitro2methylphenol	22		9.30E-01	5.90E+00	2.53E+00	1.01E+00
51285	2,4dinitrophenol	8		1.40E+00	2.10E+00	1.60E+00	2.56E-01
959988	Endosulfanalpha	14		2.30E-03	4.90E-02	1.49E-02	1.47E-02
33213659	Endosulfanbeta	20	2 / 20	7.80E-04	9.90E-02	2.65E-02	2.89E-02
1031078	Endosulfan sulfate	14		4.40E-03	9.90E-02	2.97E-02	2.96E-02
72208	Endrin	44	8 / 44	1.00E-05	9.90E-02	1.48E-02	2.36E-02
7421934	Endrin aldehyde	7		4.40E-03	1.00E-02	8.00E-03	2.43E-03
53494705	Endrin ketone	14		4.40E-03	9.90E-02	2.97E-02	2.96E-02
100414	Ethylbenzene	22	2 / 22	9.00E-04	1.50E-02	7.65E-03	5.18E-03
206440	Fluoranthene	45	45 / 45	1.30E-01	3.20E+01	4.01E+00	6.69E+00
86737	Fluorene	45	40 / 45	9.86E-03	1.40E+01	1.29E+00	2.98E+00
1024573	Heptachlor epoxide	44	4 / 44	1.00E-04	4.90E-02	9.13E-03	1.21E-02

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
76448	Heptachlor (pesticide)	42	9 / 42	5.00E-05	4.90E-02	7.69E-03	1.16E-02
591786	2Hexanone	22	1 / 22	8.00E-04	3.00E-02	1.29E-02	1.08E-02
26601649	Hexachlorobiphenyl	1	1 / 1	1.50E-04	1.50E-04	1.50E-04	
193395	Indeno(1,2,3c,d)pyrene	45	42 / 45	6.90E-02	6.30E+00	9.23E-01	1.26E+00
7439896	Iron	54	54 / 54	5.09E+03	5.82E+04	3.09E+04	1.31E+04
78591	Isophorone	22		3.80E-01	2.30E+00	6.70E-01	4.23E-01
7439921	Lead	54	54 / 54	3.90E+01	7.75E+02	1.84E+02	1.38E+02
108101	4Methyl2pentanone	22	1 / 22	5.00E-04	3.00E-02	1.35E-02	1.07E-02
7439954	Magnesium	33	33 / 33	1.05E+03	1.31E+04	3.36E+03	1.99E+03
7439965	Manganese	34	34 / 34	1.00E+02	8.00E+02	4.47E+02	1.82E+02
2245387	1,6,7Trimethylnaphthalene	9	9 / 9	5.39E-02	2.60E-01	1.52E-01	7.67E-02
7439976	Mercury	54	46 / 54	1.00E-01	2.70E+00	3.67E-01	3.85E-01
28804888	Dimethylnaphthalene	13	13 / 13	1.24E-02	4.83E-01	7.75E-02	1.24E-01
90120	1Methylnaphthalene	23	22 / 23	4.23E-03	9.80E-02	3.40E-02	2.03E-02
91576	2Methylnaphthalene	45	32 / 45	9.86E-03	1.80E+01	1.19E+00	2.92E+00
28652779	Trimethylnaphthalene	13	13 / 13	1.17E-02	4.66E-01	6.80E-02	1.21E-01
72435	Methoxychlor	14		2.30E-02	4.90E-01	1.49E-01	1.47E-01
75092	Methylene chloride	22	6 / 22	2.10E-03	8.40E-02	1.20E-02	1.71E-02
581420	2,6Dimethylnaphthalene	10	10 / 10	8.45E-03	1.45E-01	8.45E-02	4.13E-02
832699	1Methylphenanthrene	23	23 / 23	2.51E-02	2.70E-01	8.42E-02	5.52E-02
95487	2Methylphenol	22		3.80E-01	4.30E+00	1.72E+00	1.36E+00
106445	4Methylphenol	22		3.80E-01	4.30E+00	1.72E+00	1.36E+00
2385855	Mirex (pesticide = dechlorane)	30	9 / 30	1.60E-04	1.72E-02	3.12E-03	4.80E-03
	Monobutyl tin	1		1.00E-02	1.00E-02	1.00E-02	
7439987	Molybdenum	11		5.00E+00	5.05E+00	5.01E+00	1.68E-02
105679	2,4Dimethylphenol	22		3.80E-01	2.80E+00	1.28E+00	8.09E-01
2245387	2,3,5Trimethylnaphthalene	1	1 / 1	7.05E-03	7.05E-03	7.05E-03	
88744	2Nitroaniline	22		9.30E-01	2.20E+01	7.68E+00	7.34E+00
99092	3Nitroaniline	22		9.30E-01	2.20E+01	7.68E+00	7.34E+00
100016	4Nitroaniline	22		9.30E-01	2.20E+01	7.68E+00	7.34E+00

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
91203	Naphthalene	45	33 / 45	1.41E-02	6.90E+00	6.73E-01	1.33E+00
98953	Nitrobenzene	22		3.80E-01	2.30E+00	8.40E-01	4.07E-01
7440020	Nickel	34	34 / 34	1.32E+01	6.97E+01	3.56E+01	1.14E+01
621647	NnitrosodiNpropylamine	22		3.80E-01	2.30E+00	7.26E-01	4.03E-01
86306	Nnitrosodiphenylamine	22		3.80E-01	2.30E+00	8.89E-01	4.29E-01
117840	DiNoctyl phthalate	22	5 / 22	9.30E-02	2.30E+00	8.13E-01	6.28E-01
39765805	Trans nonachlor	31	31 / 31	7.90E-04	3.94E-02	2.10E-02	1.21E-02
5103731	cisNonachlor	17	15 / 17	1.54E-03	2.06E-02	1.26E-02	5.55E-03
88755	2Nitrophenol	22		3.80E-01	2.30E+00	1.03E+00	5.37E-01
100027	4Nitrophenol	22		9.30E-01	2.20E+01	7.68E+00	7.34E+00
51285	2,4Dinitrophenol	14		9.30E-01	5.90E+00	2.43E+00	1.23E+00
121142	2,4Dinitrotoluene	22		3.30E-01	2.30E+00	6.35E-01	4.41E-01
606202	2,6Dinitrotoluene	22		3.00E-01	2.30E+00	6.21E-01	4.50E-01
95476	Xylene, ortho	8	2 / 8	5.00E-04	1.40E-02	2.60E-03	4.70E-03
53190	o,p'DDD	30	26 / 30	6.90E-04	1.97E-02	6.81E-03	5.40E-03
3424826	o,p'DDE	30	10 / 30	8.00E-05	1.72E-02	2.94E-03	4.72E-03
789026	o,p'DDT	30	19 / 30	5.20E-04	1.72E-02	4.56E-03	4.54E-03
108601	2,2'Oxybis(1chloropropane)	14		3.80E-01	2.30E+00	7.83E-01	4.98E-01
1336363	PCBS, total	45	33 / 45	3.40E-02	1.20E+01	8.03E-01	1.76E+00
1825214	Pentachloroanisole	6	6 / 6	2.50E-04	1.08E-03	6.58E-04	2.90E-04
198550	Perylene	23	23 / 23	5.07E-02	6.33E-01	2.25E-01	1.47E-01
85018	Phenanthrene	45	44 / 45	8.30E-02	4.20E+01	4.65E+00	9.46E+00
108952	Phenol	22	4 / 22	2.70E-01	2.30E+00	6.90E-01	4.33E-01
7440097	Potassium	22	22 / 22	1.52E+02	2.40E+03	1.02E+03	6.51E+02
72548	p,p'DDD	44	29 / 44	4.00E-04	9.90E-02	3.10E-02	2.39E-02
72559	p,p'DDE	45	30 / 45	3.91E-03	9.90E-02	2.71E-02	2.00E-02
50293	p,p'DDT	44	20 / 44	3.70E-04	9.90E-02	1.40E-02	2.17E-02
129000	Pyrene	45	45 / 45	1.10E-01	3.60E+01	4.39E+00	8.08E+00
7782492	Selenium	34	2 / 34	2.40E-01	1.80E+00	1.01E+00	4.85E-01
7440224	Silver	23	14 / 23	5.10E-01	6.44E+01	4.50E+00	1.31E+01

Table A5-1. Descriptive Statistics for Chemicals in tidal Anacostia Sediment (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
7440235	Sodium	22	22 / 22	5.35E+01	1.69E+03	3.00E+02	3.34E+02
7440246	Strontium	11	11 / 11	9.43E+00	2.17E+01	1.54E+01	3.58E+00
100425	Styrene	22	1 / 22	4.00E-04	1.50E-02	7.19E-03	5.51E-03
156605	Trans1,2Dichloroethene	8		8.00E-04	1.20E-03	9.00E-04	1.60E-04
10061026	trans1,3Dichloropropene	22		4.00E-04	1.50E-02	6.97E-03	5.70E-03
20763886	Tributyl tin	1	1 / 1	1.00E-02	1.00E-02	1.00E-02	
127184	Tetrachlorethene	14		6.00E-03	1.50E-02	1.07E-02	3.31E-03
7440280	Thallium	22		4.60E-01	3.60E+00	1.68E+00	1.07E+00
7440315	Tin	1	1 / 1	8.64E+00	8.64E+00	8.64E+00	
108883	Toluene	22	4 / 22	5.00E-04	1.80E-02	8.21E-03	5.69E-03
	Total chlordane (alpha+cis+oxy+trans)	25	25 / 25	9.33E-03	2.26E-01	1.19E-01	6.02E-02
8001352	Toxaphene	16		4.96E-02	9.90E-01	4.13E-01	2.45E-01
7440622	Vanadium	33	33 / 33	6.90E+00	6.81E+01	3.93E+01	1.64E+01
108054	Vinyl acetate	8		1.40E-03	2.10E-03	1.64E-03	2.62E-04
75014	Vinyl chloride	22		8.00E-04	3.00E-02	1.28E-02	1.09E-02
1330207	Xylenes, total	14	1 / 14	6.00E-03	1.50E-02	1.05E-02	3.52E-03
7440666	Zinc	54	54 / 54	4.56E+01	5.12E+02	3.01E+02	1.19E+02

This table includes analytes that were omitted from the count of total analytes reported in Sections 5.2 and 6.2.5–6.2.8 (to avoid redundancy); therefore, the number of analytes in this table will differ from those reported in the aforementioned sections. Blanks in the *Det Frequency* column indicate the chemical was not detected in any of the samples. Averages and standard deviations were calculated with non-detects assigned values provided in the concentration field of the NOAA (2000) database. The averages and standard deviations do not include records where negative concentrations were assigned as missing data values to indicate detection limits that were unknown. CHEMNAME refers to the name code in the database; Det Freq, detection frequency (number of detects/number of samples); Min, minimum concentration; Max, maximum concentration; Mean, arithmetic mean; Std Dev, standard deviation of the mean; (a)E(b) refers to [$a \cong 10^b$]

Table A5-2. Descriptive Statistics for Chemicals in Water (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
309002	Aldrin	38	2 / 38	1.65E-07	2.33E-05	1.38E-06	5.21E-06
309002	Aldrin, dissolved	38		1.18E-05	1.18E-05	1.18E-05	2.51E-13
309002	Aldrin, particulate	38	2 / 38	1.65E-07	2.33E-05	1.38E-06	5.21E-06
7440382	Arsenic	33	33 / 33	2.10E-01	6.60E-01	4.43E-01	1.11E-01
22569728	Arsenic III	33	33 / 33	1.00E-02	1.80E-01	8.59E-02	5.09E-02
7440439	Cadmium	57	24 / 57	2.00E-02	4.80E-01	1.09E-01	9.41E-02
7440439	Cadmium, acid soluble	21	21 / 21	8.00E-03	9.30E-02	3.77E-02	2.13E-02
7440439	Cadmium, dissolved	57	56 / 57	1.00E-03	4.10E-02	8.75E-03	7.90E-03
	Chromium, total	57	39 / 57	6.00E-02	4.37E+00	9.74E-01	1.09E+00
118741	Hexachlorobenzene (HCB)	38	37 / 38	1.47E-07	4.07E-04	8.05E-05	7.82E-05
118741	Hexachlorobenzene (HCB), dissolved	38	31 / 38	4.01E-06	1.22E-04	4.36E-05	3.33E-05
118741	Hexachlorobenzene (HCB), particulate	38	26 / 38	1.47E-07	2.85E-04	4.06E-05	5.53E-05
7440508	Copper	57	56 / 57	1.10E-01	9.50E+00	3.64E+00	2.11E+00
7440508	Copper, acid soluble	21	21 / 21	6.90E-01	6.70E+00	2.85E+00	1.37E+00
7440508	Copper, dissolved	57	57 / 57	6.20E-01	4.02E+00	1.55E+00	6.52E-01
	Chromium, acid soluble	21	21 / 21	2.40E-01	5.59E+00	1.22E+00	1.27E+00
16065831	Chromium III	21	21 / 21	2.00E-02	1.54E+00	3.60E-01	3.50E-01
18540299	Chromium VI	21	19 / 21	1.00E-02	4.30E-01	1.49E-01	1.24E-01
	Dimethylarsenic	33	32 / 33	1.00E-02	1.19E-01	4.61E-02	2.76E-02
76448	Heptachlor (pesticide)	38	9 / 38	1.56E-07	2.85E-04	1.90E-05	5.68E-05
76448	Heptachlor (pesticide), dissolved	38	5 / 38	1.28E-06	1.81E-04	1.97E-05	3.80E-05
76448	Heptachlor (pesticide), particulate	38	7 / 38	1.56E-07	1.18E-04	9.44E-06	2.35E-05
	Mercury, dissolved	3	3 / 3	5.00E-04	9.00E-04	6.67E-04	2.08E-04
7439921	Lead	57	52 / 57	2.00E-02	9.70E+00	2.41E+00	1.86E+00
7439921	Lead, dissolved	57	57 / 57	1.20E-02	3.32E+00	6.04E-01	6.53E-01

Table A5-2. Descriptive Statistics for Chemicals in Water (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Avg (ppm)	Std Dev
22967926	Methylmercury	3	3 / 3	3.50E-05	5.80E-05	4.63E-05	1.15E-05
	Monomethylarsenic	33	32 / 33	1.00E-02	1.19E-01	3.95E-02	3.27E-02
7440020	Nickel, acid soluble	21	21 / 21	7.10E-01	5.12E+00	3.10E+00	1.06E+00
7440020	Nickel	57	56 / 57	1.50E-01	7.19E+00	2.88E+00	1.72E+00
7440020	Nickel, dissolved	57	57 / 57	4.91E-01	3.59E+00	1.74E+00	8.32E-01
39765805	Trans nonachlor	38	25 / 38	7.30E-08	7.97E-04	1.18E-04	1.97E-04
39765805	Trans nonachlor, dissolved	38	16 / 38	1.15E-06	1.25E-04	2.60E-05	3.45E-05
39765805	Trans nonachlor, particulate	38	22 / 38	7.30E-08	7.70E-04	9.66E-05	1.77E-04
7439921	Lead, acid soluble	21	21 / 21	2.50E-01	7.78E+00	3.44E+00	1.52E+00
1336363	PCBS, total	38	38 / 38	2.24E-04	1.72E-02	5.49E-03	3.33E-03
1336363	PCBS, total, dissolved	38	36 / 38	2.11E-04	4.43E-03	1.78E-03	1.02E-03
1336363	PCBS, total, particulate	38	38 / 38	1.26E-05	1.41E-02	3.73E-03	2.71E-03
72559	p,p'DDE	38	38 / 38	7.90E-06	1.45E-03	2.44E-04	2.78E-04
72559	p,p'DDE, dissolved	38	36 / 38	7.90E-06	2.18E-04	8.83E-05	4.96E-05
72559	p,p'DDE, particulate	38	34 / 38	6.00E-08	1.35E-03	1.61E-04	2.53E-04
50293	p,p'DDT	38	27 / 38	6.00E-08	6.49E-04	1.06E-04	1.66E-04
50293	p,p'DDT. Dissolved	38	7 / 38	3.32E-06	2.04E-04	1.69E-05	3.78E-05
50293	p,p'DDT. Particulate	38	26 / 38	6.00E-08	5.17E-04	9.23E-05	1.38E-04
7440666	Zinc	57	56 / 57	1.20E-01	3.18E+01	1.07E+01	8.18E+00
7440666	Zinc, dissolved	57	57 / 57	1.10E-01	1.70E+01	3.39E+00	3.85E+00
7440666	Zinc, acid soluble	21	21 / 21	1.08E+00	3.18E+01	1.40E+01	8.89E+00

This table includes analytes that were omitted from the count of total analytes reported in Sections 5.2 and 6.2.5–6.2.8 (to avoid redundancy); therefore, the number of analytes in this table will differ from those reported in the aforementioned sections. Blanks in the *Det Frequency* column indicate the chemical was not detected in any of the samples. Averages and standard deviations were calculated with non-detects assigned values provided in the concentration field of the NOAA (2000) database. The averages and standard deviations do not include records where negative concentrations were assigned as missing data values to indicate detection limits that were unknown. CHEMNAME refers to the name code in the database; Det Freq, detection frequency (number of detects/number of samples); Min, minimum concentration; Max, maximum concentration; Mean, arithmetic mean; Std Dev, standard deviation of the mean; (a)E(b) refers to [$a \cong 10^b$]

Table A5-3. Descriptive Statistics for Chemicals in Fish Tissue (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Mean (ppm)	Std Dev
83329	Acenaphthene	29	12 / 29	3.00E-04	2.83E-02	3.39E-03	5.37E-03
208968	Acenaphthylene	29	12 / 29	3.50E-04	5.00E-03	1.14E-03	9.94E-04
309002	Aldrin	32	8 / 32	1.00E-04	2.31E-03	6.67E-04	4.84E-04
120127	Anthracene	29	12 / 29	3.00E-04	1.23E-02	2.92E-03	2.66E-03
7440360	Antimony	11		2.00E-02	3.00E-02	2.55E-02	5.22E-03
11096825	Aroclor 1260	3	3 / 3	1.80E-01	4.50E-01	2.85E-01	1.45E-01
7440382	Arsenic	16	10 / 16	2.50E-02	2.66E-01	1.05E-01	9.17E-02
111444	Bis(2chloroethyl)ether	18		1.00E-03	7.00E-03	2.67E-03	1.46E-03
39638329	Bis(2chloroisopropyl) ether	18		4.00E-03	2.00E-02	8.72E-03	3.91E-03
117817	Bis(2ethylhexyl) phthalate	18	16 / 18	9.00E-03	6.40E-01	1.69E-01	1.44E-01
56553	Benz(a)anthracene	29	11 / 29	2.00E-04	5.00E-03	2.06E-03	2.19E-03
53703	Dibenz(a,h)anthracene	29	11 / 29	1.00E-05	2.00E-03	1.02E-03	8.62E-04
50328	Benzo(a)pyrene	29	11 / 29	7.00E-05	2.00E-03	9.28E-04	8.23E-04
205992	Benzo(b)fluoranthene	29	11 / 29	4.00E-05	3.00E-03	1.27E-03	1.29E-03
111911	Bis(2chloroethoxy)methane	18		5.00E-04	2.00E-03	1.47E-03	7.00E-04
192972	Benzo(e)pyrene	11	11 / 11	1.10E-04	6.40E-04	3.65E-04	1.90E-04
7440417	Beryllium	11		2.00E-02	3.00E-02	2.55E-02	5.22E-03
191242	Benzo(g,h,i)perylene	29	11 / 29	7.00E-05	5.00E-03	1.95E-03	2.26E-03
92524	Biphenyl	11	11 / 11	6.90E-04	1.06E-01	1.19E-02	3.14E-02
207089	Benzo(k)fluoranthene	29	11 / 29	6.00E-05	3.00E-03	1.23E-03	1.32E-03
101553	4Bromophenyl phenyl ether	18		6.00E-04	5.00E-03	3.08E-03	2.21E-03
118796	2,4,6Tribromophenol	18		6.00E-04	4.00E-03	2.53E-03	1.69E-03
85687	Butylbenzyl phthalate	18		1.00E-03	2.00E-01	1.12E-01	1.02E-01
92875	Benzidine	18		2.00E-03	6.00E-03	4.44E-03	1.92E-03
7440439	Cadmium	16	15 / 16	3.00E-03	2.00E-01	3.81E-02	6.67E-02
5103742	Transchlordane	12	10 / 12	3.90E-02	2.30E-01	1.38E-01	6.36E-02
5103719	Cischlordane	12	9 / 12	9.00E-02	2.30E-01	1.59E-01	5.63E-02
27304138	Oxychlordane	23	12 / 23	1.09E-03	1.00E-02	4.94E-03	3.03E-03
5103719	Chlordane alpha	29	29 / 29	2.00E-03	3.40E-01	5.65E-02	7.28E-02
5566347	Chlordane gamma	29	29 / 29	5.00E-04	9.00E-02	2.85E-02	2.69E-02
57749	Chlordane	3	3 / 3	1.50E-01	3.27E-01	2.26E-01	9.10E-02
	Chromium, total	16	10 / 16	4.00E-02	5.00E-01	1.62E-01	1.57E-01

Table A5-3. Descriptive Statistics for Chemicals in Fish Tissue (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Mean (ppm)	Std Dev
218019	Chrysene	29	11 / 29	2.60E-04	5.37E-03	2.71E-03	2.19E-03
319846	Hexachlorocyclohexanealpha	32	13 / 32	9.00E-05	8.00E-03	1.05E-03	1.40E-03
319857	Hexachlorocyclohexanebeta	29	6 / 29	2.00E-05	3.00E-03	9.78E-04	8.39E-04
319868	Hexachlorocyclohexanedelta	29	1 / 29	1.60E-04	2.00E-03	7.15E-04	4.25E-04
58899	Hexachlorocyclohexanegamma (Lindane)	32	12 / 32	1.60E-04	2.58E-03	7.68E-04	5.61E-04
59507	4Chloro3methylphenol	18		2.00E-03	2.00E-02	8.28E-03	4.65E-03
95501	1,2Dichlorobenzene	18		3.00E-04	2.00E-03	1.28E-03	8.36E-04
120821	1,2,4Trichlorobenzene	18		3.00E-04	2.00E-03	1.29E-03	8.26E-04
541731	1,3Dichlorobenzene	18		3.00E-04	2.00E-03	1.28E-03	8.36E-04
106467	1,4Dichlorobenzene	18		3.00E-04	2.00E-03	1.28E-03	8.36E-04
118741	Hexachlorobenzene (HCB)	32	13 / 32	2.50E-04	4.98E-03	2.11E-03	1.62E-03
87683	Hexachlorobutadiene	18		6.00E-04	4.00E-03	2.59E-03	1.65E-03
91941	3,3'Dichlorobenzidine	18		2.00E-03	2.00E-02	1.21E-02	9.09E-03
77474	Hexachlorocyclopentadiene	18		9.00E-04	1.00E-02	6.03E-03	4.58E-03
67721	Hexachloroethane	18		2.00E-03	1.00E-02	6.67E-03	3.90E-03
91587	2Chloronaphthalene	18		2.00E-04	1.00E-03	6.61E-04	3.93E-04
95578	2Chlorophenol	18		6.00E-04	7.00E-03	3.67E-03	2.13E-03
120832	2,4Dichlorophenol	18		4.00E-04	9.00E-03	5.67E-03	3.99E-03
87865	Pentachlorophenol	18		4.00E-03	9.00E-03	7.22E-03	2.34E-03
7440508	Copper	5	5 / 5	2.70E-01	7.50E-01	4.66E-01	2.55E-01
7005723	4Chlorophenyl phenyl ether	18		4.00E-04	2.00E-03	1.33E-03	7.82E-04
1861321	Dacthal	3	1 / 3	1.00E-03	1.00E-03	1.00E-03	0.00E+00
84662	Diethyl phthalate	18	8 / 18	1.00E-03	1.40E-02	4.00E-03	3.79E-03
132650	Dibenzothiophene	11	11 / 11	3.10E-04	1.53E-02	2.41E-03	4.32E-03
60571	Dieldrin	44	41 / 44	2.50E-04	5.20E-02	1.37E-02	1.35E-02
84742	Dinbutyl phthalate	18	17 / 18	5.00E-03	1.60E-01	6.12E-02	3.55E-02
131113	Dimethyl phthalate	18		2.00E-04	2.00E-03	1.24E-03	8.81E-04
534521	4,6dinitro2methylphenol	18		4.00E-03	1.00E-02	7.83E-03	2.83E-03
51285	2,4dinitrophenol	10		9.00E-03	9.00E-03	9.00E-03	2.40E-10
959988	Endosulfanalpha	21		5.00E-04	1.00E-03	6.29E-04	1.90E-04
33213659	Endosulfanbeta	29	2 / 29	1.50E-04	1.00E-03	4.47E-04	1.87E-04

Table A5-3. Descriptive Statistics for Chemicals in Fish Tissue (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Mean (ppm)	Std Dev
1031078	Endosulfan sulfate	18	1 / 18	8.00E-04	4.00E-03	1.09E-03	7.33E-04
72208	Endrin	32	2 / 32	1.80E-04	2.00E-03	6.67E-04	4.05E-04
7421934	Endrin aldehyde	18		7.00E-04	1.00E-03	8.83E-04	1.50E-04
206440	Fluoranthene	29	14 / 29	4.00E-04	3.06E-02	4.84E-03	7.08E-03
86737	Fluorene	29	13 / 29	3.00E-04	6.01E-02	5.21E-03	1.12E-02
1024573	Heptachlor epoxide	32	26 / 32	5.00E-04	1.70E-02	4.08E-03	4.34E-03
76448	Heptachlor (pesticide)	32	12 / 32	5.00E-05	6.10E-03	7.52E-04	1.25E-03
193395	Indeno(1,2,3c,d)pyrene	29	11 / 29	4.00E-05	2.00E+00	2.71E-01	4.90E-01
78591	Isophorone	18	1 / 18	4.00E-04	1.90E+00	1.07E-01	4.48E-01
7439921	Lead	16	16 / 16	2.50E-02	4.20E+00	4.49E-01	1.04E+00
7439965	Manganese	2	2 / 2	5.50E-01	6.00E-01	5.75E-01	3.54E-02
2245387	1,6,7Trimethylnaphthalene	11	11 / 11	8.20E-04	2.09E-01	2.68E-02	6.10E-02
7439976	Mercury	16	16 / 16	2.49E-02	1.59E-01	6.58E-02	3.30E-02
90120	1Methylnaphthalene	11	11 / 11	2.29E-03	1.84E-01	2.79E-02	5.36E-02
91576	2Methylnaphthalene	11	11 / 11	3.64E-03	2.72E-01	4.17E-02	7.96E-02
72435	Methoxychlor	21		4.00E-04	2.00E-03	1.28E-03	7.39E-04
581420	2,6Dimethylnaphthalene	11	11 / 11	1.10E-03	3.82E-01	4.45E-02	1.13E-01
832699	1Methylphenanthrene	11	11 / 11	2.50E-04	1.34E-02	2.68E-03	3.84E-03
2385855	Mirex (pesticide = dechlorane)	14	11 / 14	8.00E-05	1.00E-03	5.34E-04	3.64E-04
105679	2,4Dimethylphenol	18		7.00E-04	8.00E-03	2.75E-03	1.75E-03
91203	Naphthalene	29	25 / 29	1.00E-04	1.60E-01	2.04E-02	3.20E-02
98953	Nitrobenzene	18		1.00E-03	8.00E-03	5.50E-03	3.20E-03
7440020	Nickel	13	6 / 13	3.00E-02	7.16E-02	4.72E-02	1.35E-02
62759	Nnitrosodimethylamine	10		1.00E-02	1.00E-02	1.00E-02	1.96E-10
621647	NnitrosodiNpropylamine	18		4.00E-03	3.00E-02	1.90E-02	1.27E-02
86306	Nnitrosodiphenylamine	18		5.00E-04	1.00E-02	5.83E-03	4.80E-03
117840	DiNoctyl phthalate	18	16 / 18	7.00E-03	6.70E+00	1.65E+00	2.07E+00
39765805	Trans nonachlor	23	21 / 23	1.07E-02	3.70E-01	9.05E-02	8.84E-02
5103731	cisNonachlor	23	13 / 23	4.44E-03	8.20E-02	2.17E-02	1.99E-02
88755	2Nitrophenol	18		1.00E-03	6.00E-03	3.17E-03	1.54E-03
100027	4Nitrophenol	18		3.00E-03	2.00E-02	1.39E-02	8.01E-03
121142	2,4Dinitrotoluene	18		1.00E-03	8.00E-03	5.28E-03	3.27E-03

Table A5-3. Descriptive Statistics for Chemicals in Fish Tissue (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Mean (ppm)	Std Dev
606202	2,6Dinitrotoluene	18		1.00E-03	7.00E-03	4.39E-03	2.33E-03
3268879	Octachlorodibenzopdioxin	18	18 / 18	6.50E-07	5.71E-05	1.37E-05	1.88E-05
39001020	Octachlorodibenzofuran	18	18 / 18	1.00E-07	9.22E-05	1.49E-05	2.50E-05
53190	o,p'DDD	11	11 / 11	2.37E-03	1.43E-02	7.01E-03	4.65E-03
3424826	o,p'DDE	11	11 / 11	9.00E-05	1.27E-03	5.52E-04	4.63E-04
789026	o,p'DDT	11	10 / 11	8.00E-05	6.90E-03	2.33E-03	2.13E-03
1336363	PCBS, total	44	43 / 44	4.07E-02	4.60E+00	7.98E-01	9.63E-01
41903575	TCDD, total	18	18 / 18	5.00E-08	2.80E-06	7.50E-07	9.85E-07
36088229	PCDD, total	18	18 / 18	5.00E-08	3.90E-06	6.47E-07	9.36E-07
34465468	H6CDD, total	18	18 / 18	3.00E-07	2.34E-05	3.29E-06	5.42E-06
37871004	H7CDD, total	18	18 / 18	1.00E-07	7.30E-06	1.89E-06	1.97E-06
35822469	H7CDD1234678	18	15 / 18	1.00E-07	7.30E-06	1.89E-06	1.97E-06
39227286	H6CDD123478	18	16 / 18	1.00E-07	5.70E-06	7.97E-07	1.28E-06
57653857	H6CDD123678	18	16 / 18	5.00E-08	7.40E-06	1.42E-06	1.93E-06
40321764	PCDD12378	18	16 / 18	5.00E-08	3.90E-06	6.47E-07	9.36E-07
19408743	H6CDD123789	18	17 / 18	5.00E-08	1.03E-05	1.08E-06	2.38E-06
1746016	TCDD2378 (dioxin)	18	18 / 18	5.00E-08	2.80E-06	7.50E-07	9.85E-07
55722275	TCDF, total	18	18 / 18	5.00E-08	4.80E-06	1.01E-06	1.34E-06
30402154	PCDF, total	18	18 / 18	1.00E-07	9.75E-06	2.29E-06	2.92E-06
55684941	H6CDF, total	18	18 / 18	2.50E-07	3.21E-05	5.08E-06	7.96E-06
38998753	H7CDF, total	18	18 / 18	1.00E-07	2.20E-05	3.25E-06	5.84E-06
67562394	H7CDF1234678	18	18 / 18	5.00E-08	1.96E-05	2.42E-06	5.28E-06
70648269	H6CDF123478	18	18 / 18	5.00E-08	1.00E-05	1.59E-06	2.60E-06
55673897	H7CDF1234789	18	18 / 18	5.00E-08	2.55E-06	8.25E-07	9.11E-07
57117449	H6CDF123678	18	18 / 18	5.00E-08	8.10E-06	1.21E-06	1.96E-06
57117416	PCDF12378	18	17 / 18	5.00E-08	5.00E-06	1.09E-06	1.47E-06
72918219	H6CDF123789	18	16 / 18	1.00E-07	9.50E-06	1.47E-06	2.29E-06
60851345	H6CDF234678	18	17 / 18	5.00E-08	5.00E-06	8.08E-07	1.45E-06
57117314	PCDF23478	18	18 / 18	5.00E-08	4.75E-06	1.23E-06	1.79E-06
51207319	TCDF2378	18	18 / 18	5.00E-08	4.80E-06	1.01E-06	1.34E-06
198550	Perylene	11	11 / 11	8.00E-05	4.90E-04	2.05E-04	1.44E-04
85018	Phenanthrene	29	15 / 29	3.00E-04	1.03E-01	1.20E-02	2.18E-02

Table A5-3. Descriptive Statistics for Chemicals in Fish Tissue (sorted by chemical name)

CAS No.	CHEMNAME	Count	Det Freq	Min (ppm)	Max (ppm)	Mean (ppm)	Std Dev
108952	Phenol	18	1 / 18	1.00E-03	4.00E-02	8.78E-03	8.76E-03
72548	p,p'DDD	44	42 / 44	1.00E-03	4.80E-01	6.25E-02	8.70E-02
72559	p,p'DDE	44	43 / 44	3.70E-03	5.00E-01	9.36E-02	1.11E-01
50293	p,p'DDT	44	30 / 44	5.00E-04	5.10E-02	5.34E-03	1.00E-02
129000	Pyrene	29	14 / 29	3.00E-04	3.30E-02	4.59E-03	6.99E-03
7782492	Selenium	11	11 / 11	8.14E-02	5.04E-01	2.50E-01	1.35E-01
7440224	Silver	13	2 / 13	4.00E-03	2.50E-02	8.08E-03	7.57E-03
7440280	Thallium	11		1.00E-02	2.00E-02	1.18E-02	4.05E-03
	Total chlordane (alpha+cis+oxy+trans)	12	10 / 12	8.00E-02	8.00E-01	4.49E-01	2.16E-01
8001352	Toxaphene	3		1.00E-02	1.00E-02	1.00E-02	1.34E-10
7440666	Zinc	16	16 / 16	7.48E-01	2.37E+01	1.07E+01	6.92E+00

This table includes analytes that were omitted from the count of total analytes reported in Sections 5.2 and 6.2.5–6.2.8 (to avoid redundancy); therefore, the number of analytes in this table will differ from those reported in the aforementioned sections. Blanks in the *Det Frequency* column indicate the chemical was not detected in any of the samples. Averages and standard deviations were calculated with non-detects assigned values provided in the concentration field of the NOAA (2000) database. The averages and standard deviations do not include records where negative concentrations were assigned as missing data values to indicate detection limits that were unknown. CHEMNAME refers to the name code in the database; Det Freq, detection frequency (number of detects/number of samples); Min, minimum concentration; Max, maximum concentration; Mean, arithmetic mean; Std Dev, standard deviation of the mean; (a)E(b) refers to [$a \cong 10^b$]

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
100016	4Nitroaniline	Yes	Yes
100027	4Nitrophenol	Yes	Yes
1002535	Dibutyl tin	Yes	Yes
100414	Ethylbenzene	Yes	Yes
100425	Styrene	Yes	Yes
100516	Benzyl alcohol	Yes	Yes
10061015	cis1,3Dichloropropene	Yes	No
10061026	trans1,3Dichloropropene	Yes	No
101553	4Bromophenyl phenyl ether	Yes	Yes
1024573	Heptachlor epoxide	Yes	Yes
1031078	Endosulfan sulfate	Yes	Yes
105679	2,4Dimethylphenol	Yes	Yes
106445	4Methylphenol	Yes	Yes
106467	1,4Dichlorobenzene	Yes	Yes
106478	4Chloroaniline	Yes	Yes
107062	1,2Dichloroethane	Yes	No
108054	Vinyl acetate	Yes	No
108101	4Methyl2pentanone	Yes	Yes
108601	2,2'Oxybis(1chloropropane)	Yes	No
108883	Toluene	Yes	Yes
108907	Chlorobenzene	Yes	Yes
108952	Phenol	Yes	Yes
110758	2Chloroethylvinyl ether	Yes	No
11096825	Aroclor 1260	Yes	Yes
11097691	Aroclor 1254	Yes	Yes
11104282	Aroclor 1221	Yes	No
11141165	Aroclor 1232	Yes	No
111444	Bis(2chloroethyl)ether	Yes	Yes
111911	Bis(2chloroethoxy)methane	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
115322	Dicofol	Yes	Yes
117817	Bis(2ethylhexyl) phthalate	Yes	Yes
117840	DiNoctyl phthalate	Yes	Yes
118741	Hexachlorobenzene (HCB)	Yes	Yes
118796	2,4,6Tribromophenol	Yes	No
120127	Anthracene	Yes	Yes
120821	1,2,4Trichlorobenzene	Yes	Yes
120832	2,4Dichlorophenol	Yes	Yes
121142	2,4Dinitrotoluene	Yes	Yes
124481	Dibromochloromethane	Yes	No
12672296	Aroclor 1248	Yes	No
12674112	Aroclor 1016	Yes	No
127184	Tetrachloroethylene	Yes	No
129000	Pyrene	Yes	Yes
131113	Dimethyl phthalate	Yes	Yes
132649	Dibenzofuran	Yes	Yes
132650	Dibenzothiophene	Yes	Yes
1330207	Xylenes, total	Yes	Yes
1336363	PCBS, total	Yes	Yes
156592	cis1,2Dichlorethene	Yes	No
156605	Trans1,2Dichloroethene	Yes	No
16065831	Chromium III	Yes	Yes
1746016	TCDD2378 (dioxin)	Yes	Yes
1825214	Pentachloroanisole	Yes	Yes
18540299	Chromium VI	Yes	Yes
1861321	Dacthal	Yes	Yes
191242	Benzo(g,h,i)perylene	Yes	Yes
192972	Benzo(e)pyrene	Yes	Yes
193395	Indeno(1,2,3c,d)pyrene	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
19408743	H6CDD123789	Yes	Yes
198550	Perylene	Yes	Yes
205992	Benzo(b)fluoranthene	Yes	Yes
206440	Fluoranthene	Yes	Yes
207089	Benzo(k)fluoranthene	Yes	Yes
20763886	Tributyl tin	Yes	Yes
208968	Acenaphthylene	Yes	Yes
218019	Chrysene	Yes	Yes
2245387	2,3,5Trimethylnaphthalene	Yes	Yes
22569728	Arsenic III	Yes	Yes
22967926	Methylmercury	Yes	Yes
2385855	Mirex (pesticide = dechlorane)	Yes	Yes
26601649	Hexachlorobiphenyl	Yes	Yes
27304138	Oxychlordane	Yes	Yes
28652779	Trimethylnaphthalene	Yes	Yes
28804888	Dimethylnaphthalene	Yes	Yes
2921882	Chlorpyrifos	Yes	Yes
309002	Aldrin	Yes	Yes
319846	Hexachlorocyclohexanealpha	Yes	Yes
319857	Hexachlorocyclohexanebeta	Yes	Yes
319868	Hexachlorocyclohexanedelta	Yes	Yes
3268879	Octachlorodibenzopdioxin	Yes	Yes
33213659	Endosulfanbeta	Yes	Yes
3424826	o,p'DDE	Yes	Yes
35822469	H7CDD1234678	Yes	Yes
39001020	Octachlorodibenzofuran	Yes	Yes
39227286	H6CDD123478	Yes	Yes
39638329	Bis(2chloroisopropyl) ether	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
39765805	Trans nonachlor	Yes	Yes
40321764	PCDD12378	Yes	Yes
50293	p,p'DDT	Yes	Yes
50328	Benzo(a)pyrene	Yes	Yes
5103719	Cischlordane	Yes	Yes
5103731	cisNonachlor	Yes	Yes
5103742	Transchlordane	Yes	Yes
51207319	TCDF2378	Yes	Yes
51285	2,4Dinitrophenol	Yes	No
53190	o,p'DDD	Yes	Yes
534521	4,6dinitro2methylphenol	Yes	Yes
53469219	Aroclor 1242	Yes	No
53494705	Endrin ketone	Yes	No
53703	Dibenz(a,h)anthracene	Yes	Yes
540590	1,2Dichloroethene	Yes	No
541731	1,3Dichlorobenzene	Yes	Yes
5566347	Chlordane gamma	Yes	Yes
55673897	H7CDF1234789	Yes	Yes
55722275	TCDF, total	Yes	Yes
56235	Carbon tetrachloride	Yes	No
56553	Benz(a)anthracene	Yes	Yes
57117314	PCDF23478	Yes	Yes
57117416	PCDF12378	Yes	Yes
57117449	H6CDF123678	Yes	Yes
57125	Cyanide	Yes	Yes
57653857	H6CDD123678	Yes	Yes
57749	Chlordane	Yes	Yes
581420	2,6Dimethylnaphthalene	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
58899	Hexachlorocyclohexanegamma (Lindane)	Yes	Yes
591786	2Hexanone	Yes	Yes
59507	4Chloro3methylphenol	Yes	Yes
60571	Dieldrin	Yes	Yes
606202	2,6Dinitrotoluene	Yes	Yes
60851345	H6CDF234678	Yes	Yes
608935	Pentachlorobenzene	Yes	Yes
621647	NnitrosodiNpropylamine	Yes	Yes
62759	Nnitrosodimethylamine	Yes	No
65850	Benzoic acid	Yes	Yes
67562394	H7CDF1234678	Yes	Yes
67641	Acetone	Yes	Yes
67663	Chloroform	Yes	No
67721	Hexachloroethane	Yes	Yes
7005723	4Chlorophenyl phenyl ether	Yes	Yes
70648269	H6CDF123478	Yes	Yes
71432	Benzene	Yes	Yes
71556	1,1,1Trichloroethane	Yes	No
72208	Endrin	Yes	Yes
72435	Methoxychlor	Yes	No
72548	p,p'DDD	Yes	Yes
72559	p,p'DDE	Yes	Yes
72918219	H6CDF123789	Yes	Yes
7421934	Endrin aldehyde	Yes	No
7429905	Aluminum	Yes	Yes
7439896	Iron	Yes	Yes
7439921	Lead	Yes	Yes
7439965	Manganese	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
7439976	Mercury	Yes	Yes
7439987	Molybdenum	Yes	No
7440020	Nickel	Yes	Yes
7440224	Silver	Yes	Yes
7440246	Strontium	Yes	Yes
7440280	Thallium	Yes	No
7440315	Tin	Yes	Yes
7440360	Antimony	Yes	Yes
7440382	Arsenic	Yes	Yes
7440393	Barium	Yes	Yes
7440417	Beryllium	Yes	Yes
7440428	Boron	Yes	Yes
7440439	Cadmium	Yes	Yes
7440473	Chromium, total	Yes	Yes
7440484	Cobalt	Yes	Yes
7440508	Copper	Yes	Yes
7440622	Vanadium	Yes	Yes
7440666	Zinc	Yes	Yes
74839	Bromomethane	Yes	No
74873	Chloromethane	Yes	No
75003	Chloroethane	Yes	No
75014	Vinyl chloride	Yes	No
75092	Methylene chloride	Yes	Yes
75150	Carbon disulfide	Yes	No
75252	Bromoform	Yes	No
75274	Bromodichloromethane	Yes	No
75343	1,1Dichloroethane	Yes	No
75354	1,1Dichloroethene	Yes	No
76448	Heptachlor (pesticide)	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
77474	Hexachlorocyclopentadiene	Yes	Yes
7782492	Selenium	Yes	Yes
78591	Isophorone	Yes	Yes
78875	1,2Dichloropropane	Yes	No
789026	o,p'DDT	Yes	Yes
78933	2Butanone	Yes	Yes
79005	1,1,2Trichloroethane	Yes	No
79016	Trichloroethene	Yes	No
79345	1,1,2,2Tetrachloroethane	Yes	No
8001352	Toxaphene	Yes	No
832699	1Methylphenanthrene	Yes	Yes
83329	Acenaphthene	Yes	Yes
84662	Diethyl phthalate	Yes	Yes
84742	Dinbutyl phthalate	Yes	Yes
85018	Phenanthrene	Yes	Yes
85687	Butylbenzyl phthalate	Yes	Yes
86306	Nnitrosodiphenylamine	Yes	Yes
86737	Fluorene	Yes	Yes
86748	Carbazole	Yes	No
87683	Hexachlorobutadiene	Yes	Yes
87865	Pentachlorophenol	Yes	Yes
88062	2,4,6Trichlorophenol	Yes	Yes
88744	2Nitroaniline	Yes	Yes
88755	2Nitrophenol	Yes	Yes
90120	1Methylnaphthalene	Yes	Yes
91203	Naphthalene	Yes	Yes
91576	2Methylnaphthalene	Yes	Yes
91587	2Chloronaphthalene	Yes	Yes
91941	3,3'Dichlorobenzidine	Yes	Yes

Table A6-1. Chemicals Considered in the Human Health Screening Assessment of the Tidal Anacostia (sorted by CAS Number)

CAS No.	Analytes in NOAA (2000)	Analyzed in tidal Anacostia ?	Detected in tidal Anacostia ?
92524	Biphenyl	Yes	Yes
92875	Benzidine	Yes	No
95476	Xylene, ortho	Yes	Yes
95487	2Methylphenol	Yes	Yes
95501	1,2Dichlorobenzene	Yes	Yes
95578	2Chlorophenol	Yes	Yes
95943	1,2,4,5Tetrachlorobenzene	Yes	Yes
95954	2,4,5Trichlorophenol	Yes	Yes
959988	Endosulfanalpha	Yes	No
98953	Nitrobenzene	Yes	Yes
99092	3Nitroaniline	Yes	Yes
	Dimethylarsenic	Yes	Yes
	Monobutyl tin	Yes	No
	Monomethylarsenic	Yes	Yes
	BHCs, total	Yes	Yes

Table A6-2. Applicable or Relevant and Appropriate Criteria Considered for Fish Tissue Data

CAS	CHEMNAME	EPA: Risk10-6 (ppm)	EPA: HQ=0.1 (ppm)	FDA Guidance (ppm)	ARAR (ppm)
83329	Acenaphthene		65		65
208968	Acenaphthylene				
309002	Aldrin	0.00063	0.032	0.3	0.00063
120127	Anthracene		320		320
11096825	Aroclor 1260	0.0014	0.022	2	0.0014
7440382	Arsenic	0.0062	0.32	68	0.0062
7440382	Arsenic	0.0062	0.32	68	0.0062
117817	Bis(2ethylhexyl) phthalate	0.77	22		0.77
56553	Benz(a)anthracene	0.015			0.015
53703	Dibenz(a,h)anthracene	0.0015			0.0015
50328	Benzo(a)pyrene	0.0015			0.0015
50328	Benzo(a)pyrene	0.0015			0.0015
205992	Benzo(b)fluoranthene	0.015			0.015
192972	Benzo(e)pyrene				
191242	Benzo(g,h,i)perylene				
92524	Biphenyl		54		54
207089	Benzo(k)fluoranthene	0.15			0.15
207089	Benzo(k)fluoranthene	0.15			0.15
7440439	Cadmium		0.54	3	0.54
7440439	Cadmium		0.54	3	0.54
5103742	Transchlordane	0.0083	0.065	0.3	0.0083
5103719	Cischlordane	0.0083	0.065	0.3	0.0083
27304138	Oxychlordane				
5103719	Chlordane alpha	0.0083	0.065	0.3	0.0083
5566347	Chlordane gamma	0.0083	0.065	0.3	0.0083
5566347	Chlordane gamma	0.0083	0.065	0.3	0.0083
57749	Chlordane	0.0083	0.065	0.3	0.0083
7440473	Chromium, total		5.4	11	5.4
218019	Chrysene	1.5			1.5
319846	Hexachlorocyclohexanealpha	0.0017		0.3	0.0017
319857	Hexachlorocyclohexanebeta	0.006		0.3	0.006
319868	Hexachlorocyclohexanedelta	0.006		0.3	0.006
58899	Hexachlorocyclohexanegamma (Lindane)	0.0083	0.32	0.3	0.0083
118741	Hexachlorobenzene (HCB)	0.0067	0.86		0.0067
7440508	Copper		40		40
1861321	Dacthal		11		11
84662	Diethyl phthalate		860		860
132650	Dibenzothiophene				

60571	Dieldrin	0.00067	0.054	0.3	0.00067
60571	Dieldrin	0.00067	0.054	0.3	0.00067
84742	Dinbutyl phthalate		110		110
84742	Dinbutyl phthalate		110		110
33213659	Endosulfanbeta		6.5		6.5
1031078	Endosulfan sulfate				
72208	Endrin		0.32		0.32
206440	Fluoranthene		43		43
86737	Fluorene		43		43
1024573	Heptachlor epoxide	0.0012	0.014	0.3	0.0012
76448	Heptachlor (pesticide)	0.0024	0.54	0.3	0.0024
193395	Indeno(1,2,3c,d)pyrene	0.015			0.015
78591	Isophorone	11	220		11
7439921	Lead			1.3	1.3
7439965	Manganese		5.4		5.4
7439965	Manganese		5.4		5.4
2245387	1,6,7Trimethylnaphthalene				
7439976	Mercury		0.11	1	0.11
90120	1Methylnaphthalene				
91576	2Methylnaphthalene				
581420	2,6Dimethylnaphthalene				
832699	1Methylphenanthrene				
2385855	Mirex (pesticide = dechlorane)	0.006	0.22	0.1	0.006
91203	Naphthalene		43		43
7440020	Nickel		22	70	22
117840	DiNoctyl phthalate		22		22
39765805	Trans nonachlor				
5103731	cisNonachlor				
3268879	Octachlorodibenzopdioxin				
39001020	Octachlorodibenzofuran				
39001020	Octachlorodibenzofuran				
53190	o,p'DDD	0.045		5	0.045
3424826	o,p'DDE	0.032		5	0.032
789026	o,p'DDT	0.032	0.54	5	0.032
1336363	PCBS, total	0.0014	0.022	2	0.0014
41903575	TCDD, total				
36088229	PCDD, total				
37871004	H7CDD, total				
35822469	H7CDD1234678				
39227286	H6CDD123478				
57653857	H6CDD123678				
40321764	PCDD12378				
19408743	H6CDD123789				

1746016	TCDD2378 (dioxin)	0.000000069			0.000000069
55722275	TCDF, total				
30402154	PCDF, total				
55684941	H6CDF, total				
38998753	H7CDF, total				
67562394	H7CDF1234678				
70648269	H6CDF123478				
55673897	H7CDF1234789				
57117449	H6CDF123678				
57117416	PCDF12378				
72918219	H6CDF123789				
60851345	H6CDF234678				
57117314	PCDF23478				
51207319	TCDF2378				
198550	Perylene				
85018	Phenanthrene				
108952	Phenol		650		650
72548	p,p'DDD	0.045		5	0.045
72559	p,p'DDE	0.032		5	0.032
50293	p,p'DDT	0.032	0.54	5	0.032
129000	Pyrene		32		32
7782492	Selenium		5.4		5.4
7440224	Silver		5.4		5.4
57749	Total chlordane (alpha+cis+oxy+trans)	0.0083	0.065	0.3	0.0083
7440666	Zinc		320		320

The values shown in the table were derived from U.S. EPA. 1997. The Incidence and Severity of Sediment Contamination in Surface Waters of the United States Vol 1:National Sediment Quality Survey, Table D-1. Office of Science and Technology. EPA 823-R-97-006. Fish tissue concentration data in the NOAA 2000 database were compared to the concentration values shown in the last column of the table.

Table A6-3. Applicable or Relevant and Appropriate Criteria Considered for Water Data

CAS	CHEMNAME	Fed SWQC[1] (ppm)	DC SWQC[2] (ppm)	Md SWQC DW[3] (ppm)	Md SWQC Fish[4] (ppm)	ARAR (ppm)
309002	Aldrin	0.00013	0.00014		0.0014	0.00014
7440382	Arsenic	0.018	0.14	50	1836	0.14
22569728	Arsenic III					
7440439	Cadmium			5		5
7440473	Chromium, total			100		100
118741	Hexachlorobenzene (HCB)	0.00075	0.00077		0.0077	0.00077
7440508	Copper	1300				1300
16065831	Chromium III					
18540299	Chromium VI					
	Dimethylarsenic					
76448	Heptachlor (pesticide)	0.00021	0.00021		0.0021	0.00021
7439921	Lead			50		15
22967926	Methylmercury					
	Monomethylarsenic					
7440020	Nickel	610	4600	100	4600	4600
39765805	Trans nonachlor					
1336363	PCBS, total	0.00017	0.000045	0.5	0.0017	0.000045
72559	p,p'DDE	0.00059			0.0059	0.00059
50293	p,p'DDT	0.00059			0.0059	0.00059
7440666	Zinc	9100			69000	9100

Surface water concentration data in the NOAA 2000 database were compared to the concentration values shown in the last column of the table.

[1]Values were taken from: U.S. EPA. 1998. National Recommended Water Quality Criteria. December 10. 63(237)FR68354-68364.

[2]Values were taken from: Government of the District of Columbia, Department of Consumer and Regulatory Affairs. 1994. Water Quality Standards. March 4. 41D.C. Reg.1075.

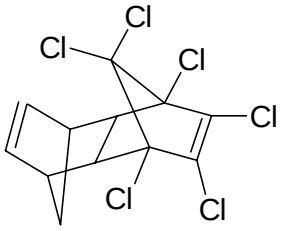
3Values were taken from: Maryland Department of the Environment. 2000. Proposed Water Quality Criteria for Toxic Substances. Available from: http://www.mde.state.md.us/wqstandards/toxics1.html-toxics8.html. June 3. Values were developed to be protective of human health via drinking water pathway.

4Values were taken from: Maryland Department of the Environment. 2000. Proposed Water Quality Criteria for Toxic Substances. Available from: http://www.mde.state.md.us/wqstandards/toxics1.html-toxics8.html. June 3. Values were developed to be protective of human health via fish consumption pathway.

APPENDIX B - TOXICITY SUMMARY

Aldrin

CHEMICAL STRUCTURE:



CAS NUMBER: 309-00-2

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.00003 mg/kg/day (IRIS; last revised 3/1/88)
Critical Effect	Liver lesions characteristic of chlorinated insecticide poisoning were observed at dose levels of 0.5 ppm and greater. These lesions were characterized by enlarged centrilobular hepatic cells, with increased cytoplasmic oxyphilia, and peripheral migration of basophilic granules.
NOAEL	None.
LOAEL	0.025 mg/kg/day estimated from dietary exposure to 0.5 ppm in rats for 2 years (The composite UF of 1000 encompasses the uncertainty of extrapolation from animals to humans, the uncertainty in the range of human sensitivities, and an additional uncertainty because the RfD is based on a LOAEL rather than a NOAEL.)
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	Kidney lesions occurred at the highest dose levels in the critical study; survival was markedly decreased at dose levels of 50 ppm and greater.

Aldrin (continued)

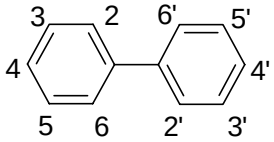
CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen
Slope Factor	17 per (mg/kg)/day (Drinking Water Unit Risk -- 4.9E-4 per (ug/L))
Human Data	Available studies concerning human carcinogenicity were inadequate.
Animal Data	Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.

Aroclor 1260

(Based on data in IRIS for Total Polychlorinated Biphenyls)

CHEMICAL STRUCTURE:



Aroclor 1260 is a mixture of 9.2% penta-, 46.9% hexa-, 36.9% hepta-, and 6.3% octachlorobiphenyl. Chlorine atoms can exist in any of the 10 positions designated.

CAS NUMBER: 11096-82-5

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish, Sediment

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

Aroclor 1260 (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen (last revised 6/1/97)
Slope Factor	For high risk sub-populations, the following slope factors for polychlorinated biphenyls were provided in IRIS: upper-bound slope factor - 2.0 per (mg/kg)/day; central-estimate slope factor - 1.0 per (mg/kg)/day. Highly exposed populations include some nursing infants and consumers of game fish, game animals, or products of animals contaminated through the food chain. The criteria for using slope factors for high risk populations include food chain exposure and sediment or soil ingestion. The reported slope factors were based on incidences of liver hepatocellular adenomas, carcinomas, cholangiomas, or cholangiocarcinomas in female Sprague-Dawley rats after dietary exposures.
Human Data	Inadequate for quantifying risk of cancer in humans after PCB exposure.
Animal Data	Increased incidences of liver adenomas and carcinomas were observed in male and female rats and thyroid adenomas or carcinomas were increased in male rats after chronic dietary exposure to Aroclor 1260.

Arsenic

CHEMICAL STRUCTURE (Bodek et al. 1988):

In nature, arsenic is associated with sulfide ores. Arsenic occurs in the environment as various inorganic and methylated acids of arsenic, exhibiting the following oxidation states: -3, 0, +3, and +5.

CAS NUMBER: 7440-38-2

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish, Sediment, Surface Water

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0003 mg/kg/day (IRIS; last revised 2/1/93)
Critical Effect	Hyperpigmentation, keratosis, and possible vascular complications.
NOAEL	0.0008 mg/kg/day based on an epidemiological study in humans (using exposure level in water was 0.009 mg/L, assumed water consumption rate of 4.5 L/day, and assumed body weight of 55 kg) (A UF of 3 is to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals).
LOAEL	0.014 mg/kg/day (using an exposure level in water of 0.17 mg/L, assumed water consumption rate of 4.5 L/day, and assumed body weight of 55 kg).
Human Data	An increased incidence of blackfoot disease that increases with age and dose was observed in humans exposed to arsenic in the drinking water. Since the high-dose group shows a clear increase in skin lesions over the low dose group, it is therefore designated a LOAEL. There is some question whether the low dose is a NOAEL or a LOAEL since there is no way of knowing what the incidence of skin lesions would be in a group where the exposure to arsenic is zero. Several other epidemiological studies found dose-related increased incidences of skin lesions.
Immunotoxicity	No data.

Arsenic (continued)

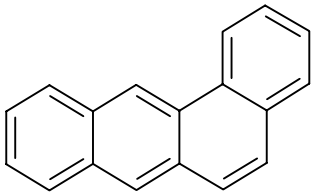
Neurotoxicity	In an epidemiological study in humans, a slight, but not statistically significant, increase in the percent of exposed individuals that have abnormal nerve conduction in the exposed population compared to control group (8/67 vs. 13/83, or 12% vs. 16%. The investigators excluded all individuals older than 47 from the nerve conduction portion of the study. These are the individuals most likely to have the longest exposure to arsenic. IRIS contends that the finding may be biologically important since it occurs in other studies at higher exposure levels. In another study in humans, abnormal electromyographic findings with increasing dose of arsenic were observed, although the sample size was extremely small.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	A; human carcinogen. Based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic.
Slope Factor	The oral slope factor is 1.5 per (mg/kg)/day (IRIS; last revised 4/10/98); the drinking water unit risk is 0.00005 per (ug/L).
Human Data	Studies of smelter worker populations, pesticide manufacturing workers, a residential population residing near a pesticide manufacturing plant, and case reports of arsenical pesticide applicators have all indicated an association between arsenic exposure and lung cancer. Increased incidence of skin cancers in humans has been associated with exposure in the drinking water, and with the therapeutic oral use of Fowler's solution (potassium arsenite). Cancers of the liver, lung, and bladder were also associated with drinking water exposures in humans.
Animal Data	Inadequate Data. There has not been consistent demonstration of carcinogenicity in test animals for various chemical forms of arsenic administered by different routes to several species.

Benz(a)anthracene

CHEMICAL STRUCTURE:



CAS NUMBER: 56-55-3

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Sediment

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

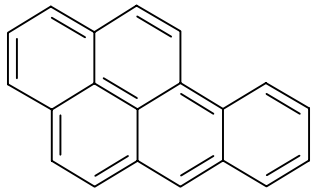
Benz(a)anthracene (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen (IRIS; last revised 3/1/94). Based on no human data and sufficient data from animal bioassays.
Slope Factor	No quantitative estimate of oral carcinogenic risk was available on IRIS for benz(a)anthracene. However, the oral slope factor used to derive screening RBCs for benz(a)anthracene is 0.73 per mg/kg/day, and is an EPA-NCEA provisional value based on a relative potency of 0.1 compared to benzo(a)pyrene (U.S. EPA, 1993). The oral slope factor for benzo(a)pyrene is 7.3 per (mg/kg)/day; (drinking water unit risk is 0.00021 per (ug/L)) (IRIS; last revised 11/1/94).
Human Data	No data.
Animal Data	Benz[a]anthracene produced tumors in mice exposed by gavage; intraperitoneal, subcutaneous or intramuscular injection, and topical application. Benz[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.

Benzo(a)pyrene

CHEMICAL STRUCTURE:



CAS NUMBER: 50-32-8

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Sediment

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

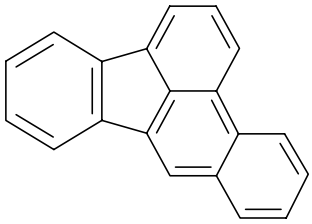
Benzo(a)pyrene (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Human data specifically linking benzo[a]pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. BAP has produced positive results in numerous genotoxicity assays.
Slope Factor	7.3 per (mg/kg)/day (IRIS; last revised 11/1/94); drinking water unit risk 0.00021 per (ug/L). The oral slope factor estimate was based on a geometric mean of four slope factors obtained by differing modeling procedures. The range of oral slope factors calculated was: 4.5 to 11.7 per (mg/kg)/day. The oral slope factor was derived from the combination of multiple data sets from two different reports using more than one sex and species.
Human Data	Inadequate. Lung cancer has been shown to be induced in humans by various mixtures of polycyclic aromatic hydrocarbons known to contain BAP including cigarette smoke, roofing tar and coke oven emissions. It is not possible, however, to conclude from this information that BAP is the responsible agent.
Animal Data	Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. BAP administered in the diet or by gavage to mice, rats and hamsters has produced increased incidences of stomach tumors. Distant site tumors have also been observed after BAP administration by various routes. Intratracheal instillation and inhalation studies in guinea pigs, hamsters and rats have resulted in elevated incidences of respiratory tract and upper digestive tract tumors. Intraperitoneal BAP injections have caused increases in the number of injection site tumors in mice and rats. Subcutaneous BAP injections have caused increases in the number of injection site tumors in mice, rats, guinea pigs, hamsters and some primates. BAP is commonly used as a positive control in many dermal application bioassays and has been shown to cause skin tumors in mice, rats, rabbits and guinea pigs. BAP is both an initiator and a complete carcinogen in mouse skin. Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal BAP exposure. BAP has also been reported to be carcinogenic in animals when administered by the following routes: i.v.; transplacentally; implantation in the stomach wall, lung, renal parenchyma and brain; injection into the renal pelvis; and vaginal painting

Benzo(b)fluoranthene

CHEMICAL STRUCTURE:



CAS NUMBER: 205-99-2

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Sediment

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

Benzo(b)fluoranthene (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen (IRIS; last revised 3/1/94). Based on no human data and sufficient data from animal bioassays. Benzo[b]fluoranthene produced tumors in mice after lung implantation, intraperitoneal (i.p.) or subcutaneous (s.c.) injection, and skin painting.
Slope Factor	No quantitative estimate of carcinogenic risk was available on IRIS based on carcinogenicity data for benzo(b)fluoranthene. However, the oral slope factor used to derive screening RBCs for benzo(b)fluoranthene is 0.73 per mg/kg/day, and is an EPA-NCEA provisional value based on a relative potency of 0.1 compared to benzo(a)pyrene (U.S. EPA, 1993). The oral slope factor for benzo(a)pyrene is 7.3 per (mg/kg)/day; (drinking water unit risk is 0.00021 per (ug/L)) (IRIS; last revised 11/1/94).
Human Data	None. Although there are no human data that specifically link exposure to benzo[b]fluoranthene to human cancers, benzo[b]fluoranthene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke.
Animal Data	In a lifetime lung implant study of benzo[b]fluoranthene exposure in 3-month-old female Osborne-Mendel rats, the incidences of epidermoid carcinomas and pleomorphic sarcomas in the lung and thorax (combined) showed a statistically significant dose-response relationship. A statistically significant increase in the incidence of liver adenomas and hepatomas (combined) occurred in mice 52 weeks after a single ip injection of benzo[b]fluoranthene. Injection site sarcomas occurred in 18/24 mice that survived three s.c. injections of benzo[b]fluoranthene over a period of 2 months. Benzo[b]fluoranthene has yielded positive results for complete carcinogenic activity and initiating activity in mouse skin-painting assays. Multiple animal studies in many species demonstrate that benzo(a)pyrene is carcinogenic in animals following administration by numerous routes. Benzo(a)pyrene has produced positive results in numerous genotoxicity assays.

Cadmium

CHEMICAL STRUCTURE:

Cd^{2+}

Cadmium is commonly associated with zinc in carbonate and sulfide ores and is also a byproduct of the refining of other metals. In the environment, cadmium exists as a free ion in freshwater(pH 6-8). However, once it enters salt water, it readily complexes with Cl^-.

CAS Number:	7440-43-9
TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999):	Non-cancer effects.
MEDIA in which RBC was EXCEEDED:	Fish.

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0005 mg/kg/day (water); 0.001 mg/kg/day (food) (IRIS; last revised 2/1/94)
Critical Effect	The highest renal cadmium concentration in humans not associated with significant proteinuria (200 ug cadmium (Cd)/gm wet human renal cortex).
NOAEL	0.005 mg/kg/day (water); 0.01 mg/kg/day (food). An uncertainty factor of 10 is used to account for intrahuman variability to the toxicity of this chemical in the absence of specific data on sensitive individuals. The choice of NOAEL does not reflect the information from any single study. Rather, it reflects the data obtained from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism and elimination. A concentration of 200 ug cadmium (Cd)/gm wet human renal cortex is the highest renal level not associated with significant proteinuria, based on data presented in the 1985 Drinking Water Criteria Document on Cadmium. A toxicokinetic model is available to determine the level of chronic human oral exposure (NOAEL) which results in 200 ug Cd/gm wet human renal cortex; the model assumes that 0.01% day of the Cd body burden is eliminated per day. Assuming 2.5% absorption of Cd from food or 5% from water, the toxicokinetic model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg/day from water and food, respectively (i.e., levels which would result in 200 ug Cd/gm wet weight human renal cortex). Thus, based on an estimated NOAEL of 0.005 mg Cd/kg/day for Cd in drinking water and an UF of 10, an RfD of 0.0005 mg Cd/kg/day (water) was calculated; an equivalent RfD for Cd in food is 0.001 mg Cd/kg/day.
LOAEL	None report in IRIS.
Human Data	No data beyond that presented for the critical effect.
Immunotoxicity	No data.

Neurotoxicity	No data.

Cadmium (continued)

Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

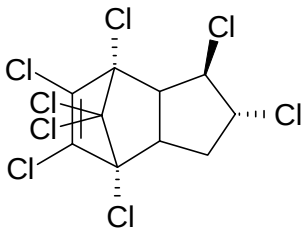
CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B1; probable human carcinogen (IRIS; last revised 6/1/92). Based on limited evidence from occupational epidemiologic studies of cadmium that is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection.
Slope Factor	No oral slope factor is available. There are no positive studies of orally ingested cadmium suitable for quantitation.
Human Data	No oral carcinogenicity data in humans was reported in IRIS.
Animal Data	Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

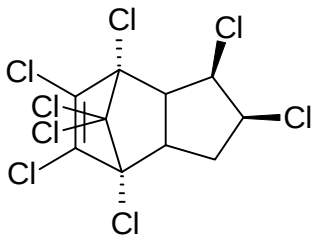
Chlordane

(The toxicology information provided below is based on data in IRIS for Technical Grade Chlordane, a mixture of chlordane isomers; no evaluations were available on IRIS (U.S. EPA, 2000a) or in the HEAST (U.S. EPA, 1997a) concerning the toxicity of individual chlordane isomers)

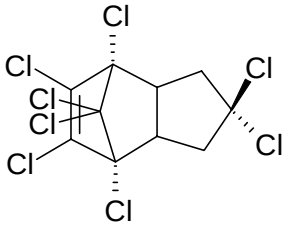
CHEMICAL STRUCTUREs:



beta-Chlordane
CAS 5103-71-9



alpha-Chlordane
CAS 5103-71-9



gamma-Chlordane
CAS 5566-34-7

CAS NUMBERs: 57-74-9 (technical)
5103-71-9 (alpha, cis)
5103-74-2 (beta, trans)
5566-34-7 (gamma, trans)

TOXICOLOGICAL BASIS for
RBC (U.S. EPA, 1999) or ARAR (U.S. EPA, 1997b): Cancer effects

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0005 mg/kg/day (IRIS; last revised 2/7/98)
Critical Effect	Hepatic necrosis in a 2-year oral exposure assay in mice.
NOAEL	0.15 mg/kg-day. The following uncertainty factors are applied to the NOAEL derived from the principal study: 10 for consideration of intraspecies variation, 10 for consideration of interspecies extrapolation, and 3 for lack of any reproductive studies.
LOAEL	0.75 mg/kg-day

Human Data	Available occupational studies, although limited, give no indication that the liver is a target organ in humans as a consequence of chronic exposure to low levels of chlordane. Recent epidemiological findings indicate that neurotoxicity may be a relevant human toxicological endpoint as a consequence of chronic as well as acute chlordane exposure. Neurotoxicity and possibly hematotoxicity are the principal endpoints of acute chlordane toxicity in both experimentally poisoned animals and accidentally poisoned humans, with tremors and convulsions being common interspecies symptoms.

Chlordane (continued)

Immunotoxicity	Several investigations in laboratory animals have assessed the effects of chlordane on the immunological system of offspring exposed during gestation and found that chlordane may affect cell-mediated immunity.
Neurotoxicity	Neurotoxicity endpoints are the principal endpoints of acute chlordane toxicity in both experimentally poisoned animals and accidentally poisoned humans, with tremors and convulsions being common interspecies symptoms. Adults (109 women and 97 men) who had been exposed to uncertain levels of chlordane via both air and oral routes were examined. Significant ($p < 0.05$) differences were observed with a battery of neurophysiological and neuropsychological function tests. Also, profiles of mood states (including tension, depression, anger, vigor, fatigue, and confusion) all were affected significantly ($p < 0.0005$), as compared to a referent population. These results indicate that neurological effects are a relevant endpoint in humans exposed to chlordane.
Reproductive Toxicity	No multi-generational reproductive studies, by any route, exist for technical chlordane.
Developmental Toxicity	Behavioral changes were observed in pups of mice exposed orally by gavage on gestation days 12-19. Several investigations in laboratory animals have assessed the effects of chlordane on the immunological system of offspring exposed during gestation and found that chlordane may affect cell-mediated immunity.
Other Systemic Toxicity	Case studies of aplastic anemia are associated with acute exposure to chlordane implicating this pesticide in bone marrow toxicity. Bone marrow hematopoietic activity in mice, as measured by the ability of bone marrow cells to undergo clonal expansion in response to stimulating factors, and spleen colony forming units (after irradiation) both showed a significant dose-related depression.

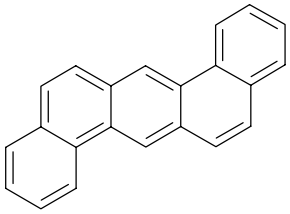
Chlordane (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen, based on (1) human epidemiology studies showing non-Hodgkin's lymphoma in farmers exposed to chlordane and case reports of aplastic anemia (chlordane associated with home use are inadequate to demonstrate carcinogenicity); (2) animal studies in which benign and malignant liver tumors were induced in both sexes of four strains of mice and occurred with an elevated, but not statistically significant, incidence in a fifth strain (liver toxicity but no tumors was observed in rats of two strains); and (3) structural similarity to other rodent liver carcinogens.
Slope Factor	0.35 per mg/(kg-day) (IRIS; last revised 2/7/98); drinking water unit risk 0.00001 per (ug/L).
Human Data	Inadequate to quantify a oral carcinogenicity dose-response.
Animal Data	Available data were sufficient to derive an oral slope factor. Chlordane treatment has induced benign or malignant liver tumors in each of five strains of mice in which bioassays have been reported.

Dibenz(a,h)anthracene

CHEMICAL STRUCTURE:



CAS NUMBER: 53-70-3

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Sediment

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

Dibenz(a,h)anthracene (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

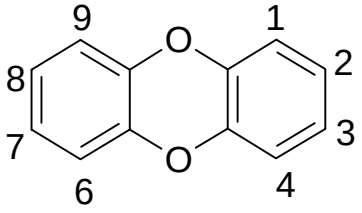
Weight-of-evidence	B2; probable human carcinogen (IRIS; last revised on 3/1/94). Based on no human data and sufficient data from animal bioassays. Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenz[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.
Slope Factor	No quantitative estimate of carcinogenic risk was available on IRIS based on carcinogenicity data for dibenz(a,h,)anthracene. However, the oral slope factor used to derive screening RBCs for dibenz(a,h)anthracene is 7.3 per mg/kg/day, and is an EPA-NCEA provisional value based on a relative potency of 1 compared to benzo(a)pyrene (U.S. EPA, 1993). The oral slope factor for benzo(a)pyrene is 7.3 per (mg/kg)/day; (drinking water unit risk is 0.00021 per (ug/L)) (IRIS; last revised 11/1/94).
Human Data	None. Although there are no human data that specifically link exposure to dibenz[a,h]anthracene with human cancers, dibenz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke.
Animal Data	Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenz[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures. Multiple animal studies in many species demonstrate that benzo(a)pyrene is carcinogenic in animals following administration by numerous routes. Benzo(a)pyrene has produced positive results in numerous genotoxicity assays.

Dibenzo-p-Dioxins and Dibenzofurans

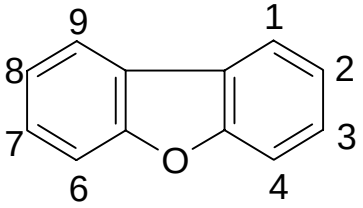
(HxCDD, PxCDD, TxCDD, HxCDF, PxCDF, and TxCDF)

CHEMICAL STRUCTURE:

Dibenzo-p-dioxins (CDDs):



Dibenzofurans (CDFs):



Individual CDD and CDF molecules are specified according to the number and position of chlorine atoms in the molecule. For instance, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) has one chlorine at each of the 2, 3, 7, and 8 positions. CDDs and CDFs with chlorine substitutions in at least the 2, 3, 7, and 8 positions are thought to have dioxin-like toxicity, and are evaluated in risk assessment in terms of 2,3,7,8-TCDD equivalent concentrations.

CAS NUMBER (2,3,7,8-TCDD): 1746-01-6

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.

Other Systemic Toxicity	No data.

Dibenzo-p-Dioxins and Dibenzofurans (continued)

CARCINOGENICITY (U.S. EPA, 1997a; no data available on IRIS for 2,3,7,8-TCDD)

Weight-of-evidence	B2 (HEAST; basis was not reported); oral unit risk 4.5 ug/L.
Slope Factor	An oral slope factor of 150,000 per mg/kg/day was reported in HEAST for 2,3,7,8-TCDD, but details concerning the critical study and other relevant data were not reported (U.S. EPA, 1997a). No quantitative estimate of carcinogenic risk for 2,3,7,8-TCDD was available on IRIS. 2,3,7,8-TCDD is thought to be the most potent toxin among the 30 or so dioxin-like compounds. Dioxin-like compounds are often found in complex mixtures, thus a toxicity equivalency procedure was developed by the U.S. EPA to describe the cumulative toxicity of these mixtures (U.S. EPA, 2000b). Toxicity equivalency factors (TEFs) were developed for dioxin-like chemicals based on their toxicity relative to 2,3,7,8-TCDD. For risk assessment of dioxin mixtures, a potency-adjusted concentration of each compound is calculated by multiplying its concentration by its TEF (referred to as its toxic equivalency (TEQ) concentration), and the individual TEQs are summed to provide an estimated total 2,3,7,8-TCDD equivalent exposure point concentration (U.S. EPA, 1989, 2000b). The U.S. EPA (1989) provides more detailed guidance for applying the TEFs. TEFs for dioxin-like COPCs were provided in U.S. EPA (2000b), and are presented below in Table B1. For the screening human health risk assessment, individual RBCs of dioxin-like compounds were determined by dividing the RBC for 2,3,7,8-TCDD by the TEF for the compound, and then comparing against the unadjusted maximum concentration of the compound in the medium. This procedure is mathematically equivalent to first multiplying the maximum concentration for the individual compound by the corresponding TEF and then comparing to the unadjusted RBC for 2,3,7,8-TCDD. An exception to the rule of screening dioxin-like chemicals using the TEF approach was 1,2,3,7,8,9-H6CDD, which has its own oral slope factor of 6200 per mg/kg/day reported in IRIS (last revised on 3/1/91) (U.S. EPA, 2000a). The individual RBC reported in U.S. EPA (1993) for 1,2,3,7,8,9-H6CDD (CAS No.19408-74-3) was used instead of applying the TEF for this chemical from U.S. EPA (1989).
Human Data	No human data were reported in HEAST (U.S. EPA, 1997a) or IRIS (U.S. EPA, 2000a).
Animal Data	The oral slope factor for 2,3,7,8-TCDD was derived based on liver tumors in rats after chronic dietary exposure. No other data were reported in IRIS or HEAST.

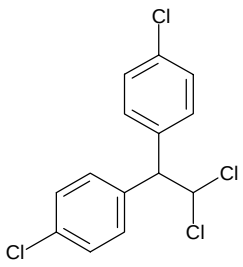
Dibenzo-p-Dioxins and Dibenzofurans (continued)

Table B1. TEF values for the dioxin-like compounds that were COPCs in fish in the screening level human health risk assessment (U.S. EPA, 2000b).

Compound	CAS Number	TEF	
	Dibenzo-p-dioxins		
2,3,7,8-TCDD	1746-01-6	1	
1,2,3,7,8-PCDD	40321-76-4	0.5	
1,2,3,4,7,8-H6CDD	39227-28-6	0.1	
1,2,3,6,7,8-H6CDD	57653-85-7	0.1	
1,2,3,7,8,9-H6CDD	19408-74-3	0.1	(not used in the COPC screening)
1,2,3,4,6,7,8-H7CDD	35822-46-9	0.01	
Octachlorodibenzo-p-dioxin	3268-87-9	0.001	
	Dibenzofurans		
2,3,7,8-TCDF	51207-31-9	0.1	
1,2,3,7,8-PCDF	57117-41-6	0.05	
2,3,4,7,8-PCDF	57117-31-4	0.5	
1,2,3,4,7,8-H6CDF	70648-26-9	0.1	
1,2,3,6,7,8-H6CDF	57117-44-9	0.1	
1,2,3,7,8,9-H6CDF	72918-21-9	0.1	
2,3,4,6,7,8-H6CDF	60851-34-5	0.1	
1,2,3,4,6,7,8-H7CDF	67562-39-4	0.01	
1,2,3,4,7,8,9-H7CDF	55673-89-7	0.01	
Octachlorodibenzofuran	390001-02-0	0.001	

Dichlorodiphenyldichloroethane *(p,p'*-DDD)

CHEMICAL STRUCTURE:



CAS NUMBER: 72-54-8

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

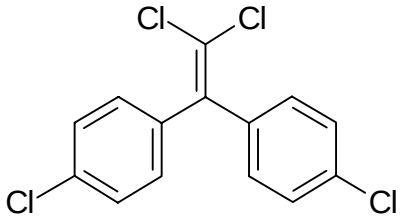
Dichlorodiphenyldichloroethane (p,p'-DDD) (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Based on an increased incidence of lung tumors in male and female mice, liver tumors in male mice and thyroid tumors in male rats. DDD is structurally similar to, and is a known metabolite of DDT, a probable human carcinogen.
Slope Factor	0.24 /mg/kg/day (IRIS; last revised 8/22/88); drinking water unit risk 0.0000069 /ug/L.
Human Data	None. Human epidemiological data are not available for DDD. Evidence for the carcinogenicity in humans of DDT, a structural analog, is based on autopsy studies relating tissue levels of DDT to cancer incidence. These studies have yielded conflicting results.
Animal Data	The oral slope factor was derived based on liver tumors in mice observed after chronic dietary exposure. Increased incidence of thyroid tumors (follicular cell adenomas and carcinomas) were observed in male rats fed DDD in the diet for 103 weeks suggesting a possible carcinogenic effect, but the incidence did not appear to be dose-related. DDD is structurally similar to, and is a metabolite of, DDT, a probable human carcinogen, in rats, mice, and humans. Positive effects were found with DDD in mammalian cytogenetic assays and a host-mediated assay.

Dichlorodiphenyldichloroethylene (*p,p'*-DDE)

CHEMICAL STRUCTURE:



CAS NUMBER: 72-55-9

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish
MEDIA in which ARAR (U.S. EPA, 1998) was EXCEEDED: Surface Water

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

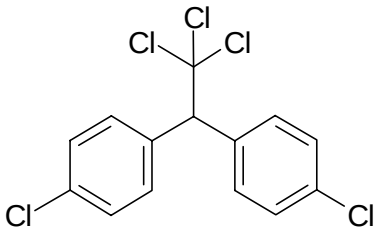
Dichlorodiphenyldichloroethylene (p,p'-DDE) (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Based on increased incidence of liver tumors including carcinomas in two strains of mice and in hamsters and thyroid tumors in female rats exposed in the diet.
Slope Factor	3.4E-1/mg/kg/day (IRIS; last revised on 8/22/88); drinking water unit risk -- 9.7E-6/ug/L.
Human Data	None. Human epidemiological data are not available for DDE. Evidence for the human carcinogenicity of DDT, a structural analog, is based on autopsy studies relating tissue levels of DDT to cancer incidence. These studies have yielded conflicting results.
Animal Data	The oral slope factor for *p,p'*-DDE was derived from dose-response data on the incidence of hepatocellular carcinomas in one strain of mice, hepatomas in another mouse strain, and hepatomas in hamsters after chronic dietary exposures. The oral quantitative estimate is a geometric mean of six slope factors computed from incidence data by sex from the three studies. Dietary exposure in female rats induced a significant dose-dependent trend in the incidence of thyroid tumors, but the Fisher exact test was not significant. DDE was mutagenic in mouse lymphoma (L5178Y) cells and chinese hamster (V79) cells, but not in Salmonella.

Dichlorodiphenyltrichloroethane (*p,p'*-DDT)

CHEMICAL STRUCTURE:



CAS NUMBER: 50-29-3

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish
MEDIA in which ARAR (U.S. EPA, 1998) was EXCEEDED: Surface Water

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0005 mg/kg/day (IRIS; last revised on 2/1/96)
Critical Effect	Liver lesions in a 27-week rat feeding study. Weanling rats (25/sex/group) were fed commercial DDT (81% P,P isomer and 19% O,P isomer) at levels of 0, 1, 5, 10 or 50 ppm for 15-27 weeks. Increasing hepatocellular hypertrophy, especially centrilobularly, increased cytoplasmic oxyphilia, and peripheral basophilic cytoplasmic granules (based on H and E paraffin sections) were observed at dose levels of 5 ppm and above. The effect was minimal at 5 ppm (LOAEL) and more pronounced at higher doses. DDT fed to rats for 2 years caused liver lesions at all dose levels (10-800 ppm of diet). DDT-induced liver effects were observed in mice, hamsters and dogs as well.
NOAEL	0.05 mg/kg/day (1 ppm in the diet). A factor of 10 each was applied for the uncertainty of interspecies conversion and to protect sensitive human subpopulations. An uncertainty factor for subchronic to chronic conversion was not included because of a corroborating chronic study in the data base.
LOAEL	5 ppm in the diet
Human Data	No data reported in IRIS.
Immunotoxicity	No data reported in IRIS.
Neurotoxicity	No data reported in IRIS.

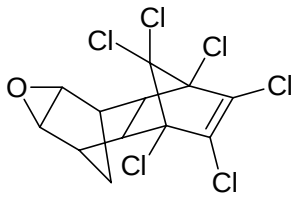
Reproductive Toxicity	In one 3-generation rat reproduction study, offspring mortality increased at all dose levels, the lowest of which corresponds to about 0.2 mg/kg bw/day. Three other reproduction studies (rat and mouse) show no reproductive effects at much higher dose levels.

Dichlorodiphenyltrichloroethane (p,p'-DDT) (continued)

Developmental Toxicity	In the critical study, weanling rats (25/sex/group) were fed commercial DDT (81% p,p isomer and 19% o,p isomer) at levels of 0, 1, 5, 10 or 50 ppm for 15-27 weeks. No interference with growth was noted at any level.
Other Systemic Toxicity	No other effects were observed in the critical study.

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Based on the observation of tumors (generally of the liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE.
Slope Factor	0.34 per mg/kg/day (IRIS; last revised on 5/1/91); drinking water unit risk - 0.0000097 per (ug/L). Ten sex-specific slope factors were derived from six studies based on dose-response data for either benign or malignant liver tumors in rats or mice after dietary exposures; all 10 slope factors fell within a 13-fold range. A geometric mean of the 10 slope factors was used for the overall slope factor of 3.4E-1. The slope factor derived from the mouse data alone was 4.8E-1 while that derived from the rat data alone was 1.5E-1. There was no apparent difference in slope factor as a function of sex of the animals. The geometric mean of the slope factors from the mouse and rat data combined was identical for the same tumor site as that for DDE [3.4E-1 per (mg/kg)/day], a structural analog.
Human Data	The existing epidemiological data are inadequate to quantify a dose-response relationship. Autopsy studies relating tissue levels of DDT to cancer incidence have yielded conflicting results. Studies of occupationally exposed workers and volunteers have been of insufficient duration to be useful in assessment of the carcinogenicity of DDT to humans.



Animal Data	Twenty-five animal carcinogenicity assays have been reviewed for DDT. Nine feeding studies, including two multigenerational studies, have been conducted in the following mouse strains: BALB/C, CF-1, A strain, Swiss/Bombay and (C57B1)x(C3HxAkR). Only one of these studies, conducted for 78 weeks, showed no indication of DDT tumorigenicity. Both hepatocellular adenomas and carcinomas were observed in six mouse liver tumor studies. Both benign and malignant lung tumors were observed in two studies wherein mice were exposed both in utero and throughout their lifetime. Doses producing increased tumor incidence ranged from 0.15-37.5 mg/kg/day. Three studies using Wistar, MRC Porton and Osborne-Mendel rats and doses from 25-40 mg/kg/day produced increased incidence of benign liver tumors. Another study wherein Osborne-Mendel rats were exposed in this dietary dose range for 78 weeks was negative as were three additional assays in which lower doses were given. Tests of DDT in hamsters have not resulted in increased tumor incidence. Unlike mice and humans, hamsters accumulate DDT in tissue but do not metabolize it to DDD or DDE. Studies of DDT in dogs and monkeys have not shown a carcinogenic effect. However, the length of these studies (approximately 30% of the animals' lifetimes) was insufficient to assess the carcinogenicity of DDT. DDT has been shown to produce hepatomas in trout.

Dieldrin

CHEMICAL STRUCTURE:

CAS NUMBER:	60-57-1
TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999):	Cancer effects.
MEDIA in which RBC was EXCEEDED:	Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.00005 mg/kg/day (IRIS; last revised 9/1/90)
Critical Effect	Liver lesions in a 2-year rat feeding study. At the end of 2 years, females fed 1.0 and 10.0 ppm (0.05 and 0.5 mg/kg/day) had increased liver weights and liver-to-body weight ratios ($p<0.05$). Histopathological examinations revealed liver parenchymal cell changes including focal proliferation and focal hyperplasia.
NOAEL	0.005 mg/kg/day (0.1 ppm in the diet). The UF of 100 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans and uncertainty in the threshold for sensitive humans.
LOAEL	0.05 mg/kg/day (1 ppm in the diet)
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	In the principle study in rats, at 10.0 ppm (0.5 mg/kg/day) all animals became irritable and exhibited tremors and occasional convulsions. Convulsions were also reported in dogs at 0.5 mg/kg/day in a 2-year feeding study.
Reproductive Toxicity	No data.
Developmental Toxicity	The results from a mouse developmental toxicity study were presented only in the following summary form in IRIS: teratogenic NOEL=6.0 mg/kg/day, gestational days 7-16); maternal LEL=6.0 mg/kg/day, decrease in maternal weight gain); fetotoxic LEL=6.0 mg/kg/day, decreased numbers of caudal ossification centers and increases in supernumerary ribs).

Dieldrin
(continued)

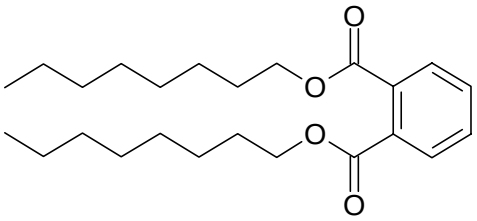
Other Systemic Toxicity	In the principle study, body weight, food intake, and general health remained unaffected throughout the 2-year feeding exposure period, although at 10.0 ppm (0.5 mg/kg/day) all rats became irritable and exhibited tremors and occasional convulsions. No effects were seen in various hematological and clinical chemistry parameters. Hepatomegaly and liver histopathologies were seen in rats in two chronic oral assays. Increased liver weight and liver/body weight ratios, increased plasma alkaline phosphatase, decreased serum protein concentration, weight loss, and convulsions were seen in dogs in two 2-year feeding studies.

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.
Slope Factor	16 per (mg/kg)/day (IRIS; last revised 7/1/93); drinking water unit risk 0.00046 per (ug/L).
Human Data	Available data were inadequate to quantify an oral carcinogenicity dose-response. Two studies of workers exposed to aldrin and to dieldrin reported no increased incidence of cancer.
Animal Data	Dieldrin has been shown to be carcinogenic in various strains of mice of both sexes. At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation confirmation, to pulmonary metastases. The slope factor is the geometric mean of 13 slope factors calculated from liver carcinoma data in both sexes of several strains of mice. Inspection of the data indicated no strain or sex specificity of carcinogenic response. Seven studies with four strains of rats fed 0.1 to 285 ppm dieldrin varying in duration of exposure from 80 weeks to 31 months did not produce positive results for carcinogenicity

Di-n-octyl phthalate

CHEMICAL STRUCTURE:



CAS NUMBER: 117-84-0

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Non-cancer effects.

MEDIA in which RBC or ARAR was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA,1997a)

RfD	0.02 mg/kg/day (HEAST; U.S. EPA, 1997a).
Critical Effect	Increased kidney and liver weights and increased levels of liver enzymes SGOT and SGPT in serum were critical effects in rats fed diets containing di-n-octyl phthalate for 7 to 12 months.
NOAEL	Not reported in HEAST.
LOAEL	175 mg/kg/day. An uncertainty factor of 1000 was reported applied, but the basis for the uncertainty factor was not provided.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

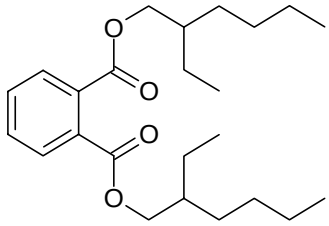
CARCINOGENICITY (U.S. EPA, 1997a, 2000a)

Weight-of-evidence	No data.

Slope Factor	No data.
Human Data	No data.
Animal Data	No data.

bis(2-Ethylhexyl) phthalate

CHEMICAL STRUCTURE:



CAS NUMBER: 117-81-7

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.02 mg/kg/day (IRIS; last revised on 5/1/91)
Critical Effect	Increased relative liver weight.
NOAEL	None.
LOAEL	0.04% of diet (19 mg/kg bw/day) in a guinea pigs subchronic-to-chronic oral bioassay (Factors of 10 each were used for interspecies variation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD is set on a LOAEL, the effect observed was considered to be minimally adverse.)
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	In a 2-year dietary exposure study in rats, no effect of treatment on either parental or F1 group was seen with respect to mortality, life expectancy, hematology, or histopathology of organs. Both parental and F1 rats high exposure groups were retarded in growth and had increased kidney and liver weights.

Other Systemic Toxicity	No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen, or testes in the guinea pig subchronic-to-chronic oral bioassay. In a 2-year dietary exposure study in rats, no effect of treatment on either parental or F1 group was seen with respect to mortality, life expectancy, hematology, or histopathology of organs. Both parental and F1 rats high exposure groups were retarded in growth and had increased kidney and liver weights.

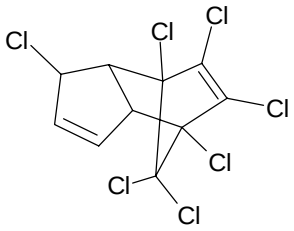
bis(2-Ethylhexyl) phthalate (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Based on the finding that orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.
Slope Factor	Oral slope factor: 0.014 per mg/kg/day (IRIS; last revised 2/1/93); drinking water unit risk 4.0E-7 per (ug/L)
Human Data	Inadequate for quantifying cancer risk in humans.
Animal Data	A statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice in a 2-year dietary exposure assay. The combined incidence of neoplastic nodules and hepatocellular carcinomas was statistically significantly increased in the high-dose male rats. A positive dose response trend was also noted.

Heptachlor (pesticide)

CHEMICAL STRUCTURE:



CAS NUMBER: 76-44-8

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish
MEDIA in which ARAR (GDC, 1994) was EXCEEDED: Surface Water

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0005 mg/kg/day (IRIS; last revised 3/1/91)
Critical Effect	Liver weight increases in male rats in a 2-year feeding study.
NOAEL	0.15 mg/kg/day (3 ppm in diet) (Reported in IRIS as a NOEL). Based on a chronic exposure study, an uncertainty factor of 100 was used to account for inter- and intraspecies differences. An additional factor of 3 was considered appropriate because of the lack of chronic toxicity data in a second species, for a total uncertainty factor of 300. The serious deficiencies in the toxicologic data base would normally warrant a 10-fold factor for this area of uncertainty. However, toxicity data for other cyclodiene insecticides (aldrin, dieldrin, chlordane, and heptachlor epoxide) suggest that dogs and rats do not differ greatly in sensitivity to the effects of this class of compounds. Furthermore, liver toxicity has been fairly well established as the most sensitive endpoint for this class of compounds, which reduces the uncertainty attributable to the lack of information on other toxic effects.
LOAEL	0.25 mg/kg/day (5 ppm in diet) (Reported in IRIS as an LEL).
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	There were several case reports involving central nervous system effects and neuroblastomas in children with pre- or postnatal exposure to chlordane and heptachlor.

Reproductive Toxicity	No detailed study descriptions were provided in IRIS; the following data were presented in summary form only. The results of a feeding 1-generation reproduction in rats are as follows: NOEL=5 ppm (0.25 mg/kg/day); LEL=7 ppm (0.35 mg/kg/day) (increased pup death). The results of a 3-generation reproduction study in rats are as follows: NOEL=10 ppm (0.5 mg/kg/day) (no adverse effects).

Heptachlor
(continued)

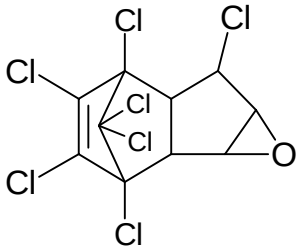
Developmental Toxicity	No data.
Other Systemic Toxicity	There were several case reports involving blood dyscrasias in children with pre- or postnatal exposure to chlordane and heptachlor. The following summary of results was reported in IRIS for an 8-month feeding study in rats: NOEL=none; LEL=5 ppm (0.25 mg/kg/day) (swelling of cells).

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Sufficient evidence of carcinogenicity in animals exist from studies in which benign and malignant liver tumors were induced in three strains of mice of both sexes. Several structurally related compounds are liver carcinogens.
Slope Factor	4.5 per (mg/kg)/day (IRIS; last revised 7/1/93); drinking water unit risk -- 0.00013 per (ug/L)
Human Data	Data from 11 case studies and 3 epidemiological studies were inadequate to quantify an oral carcinogenicity dose-response.
Animal Data	The oral slope factor was derived based on the dose-response data of hepatocellular carcinomas in two feeding studies in mice. No indication of treatment-related increase of tumors has been reported in chronic studies with rats.

Heptachlor epoxide

CHEMICAL STRUCTURE:



CAS NUMBER: 1024-57-3

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.000013 mg/kg/day (IRIS; last revised 3/1/91)
Critical Effect	Increased liver-to-body weight ratio in male and female dogs in a 60-day feeding study
NOAEL	None. (Reported as a NOEL in IRIS)
LOAEL	0.0125 mg/kg/day (0.5 ppm in the diet) (Reported as an LEL in IRIS). An uncertainty factor of 1000 was used to account for inter- and intraspecies differences and to account for the fact that a NOEL was not attained.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No detailed study descriptions were provided in IRIS; the following data were presented in summary form only. The results of a 2-generation reproduction in dogs are as follows: NOEL=1 ppm (0.025 mg/kg/day); LEL=3 ppm (0.075 mg/kg/day) (liver lesions in pups); Reproductive NOEL=5 ppm (0.125 mg/kg/day); reproductive LEL=7 ppm (0.175 mg/kg/day) (pup survival). The results of a 3-generation reproduction in rats are as follows: NOEL=5 ppm (0.25 mg/kg/day); LEL=10 ppm (0.5 mg/kg/day) (pup mortality).
Developmental Toxicity	No data.
Other Systemic Toxicity	Results of a 2-year feeding study in rats were summarized in IRIS as follows: LEL=0.5 ppm (0.025 mg/kg/day) (females - vacuolar changes in central hepatic lobule); NOEL not established.

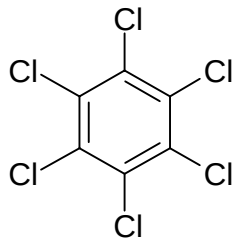
Heptachlor epoxide (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Sufficient evidence exists of the carcinogenicity of heptachlor epoxide in animals from rodent studies in which liver carcinomas were induced in two strains of mice of both sexes and in CFN female rats. Several structurally related compounds are liver carcinogens.
Slope Factor	9.1 per (mg/kg)/day (IRIS; last revised 7/1/93); drinking water unit risk 0.00026 per (ug/L).
Human Data	Inadequate. There are no published epidemiologic evaluations of heptachlor epoxide.
Animal Data	Four long-term cancer bioassays of heptachlor epoxide have been reported. The major finding in mice has been an increased incidence of liver carcinomas. The oral slope factor was derived based on the dose-response data of hepatocellular carcinomas in two feeding studies in mice. Analyses of bioassay data with rats reported a significant increase of hepatic carcinomas above the controls in the female rats and a significant increase of hepatic nodules in the males over the controls.

Hexachlorobenzene

CHEMICAL STRUCTURE:



CAS NUMBER: 118-74-1

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0008 mg/kg/day (IRIS; last revised 4/1/91)
Critical Effect	The 8.0-ppm F1 groups were reported to have an increase ($p<0.05$) in hepatic centrilobular basophilic chromogenesis. At 40 ppm, the F1 groups showed increases ($p<0.05$) in pup mortality, hepatic centrilobular basophilic chromogenesis, and severe chronic nephrosis (males only).
NOAEL	0.08 mg/kg/day (1.6 ppm in diet). An uncertainty factor of 100 was applied; 10 for interspecies and 10 for intraspecies variability.
LOAEL	0.29 mg/kg/day (8.0 ppm in diet)
Human Data	The toxicity of long-term dietary exposure of humans to hexachlorobenzene was demonstrated by the epidemic of porphyria cutanea tarda (PCT) in Turkish citizens who accidentally consumed bread made from grain treated with hexachlorobenzene. In children less than 1 year of age, pink sore disease was observed along with 95% mortality. In addition to the PCT-associated symptoms of skin lesions, hypertrichosis, and hyperpigmentation, the exposure caused neurotoxicity and liver damage. Follow-up studies reported PCT symptoms, reduced growth and arthritic changes in the appendages of children who were directly or indirectly (i.e., through breast milk) exposed. These human data cannot be used for quantitative risk assessment purposes because accurate exposure data (dose and duration) are lacking.
Immunotoxicity	No data.
Neurotoxicity	No data.

Reproductive Toxicity	An extensive number of animal research studies have been conducted on hexachlorobenzene including reproductive studies, but they were not summarized in IRIS.

Hexachlorobenzene (cont)

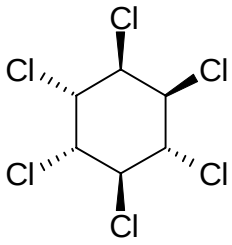
Developmental Toxicity	The derivation of the oral RfD is based on a 130-week, multigeneration rat study in which the males and females in the F0 generation were fed diets containing hexachlorobenzene (analytical grade) for 90 days prior to mating and until 21 days after parturition (at weaning). The F1 animals were exposed to hexachlorobenzene and metabolites in utero, from maternal nursing and from their diets for the remainder of their lifetime (130 weeks). Increased mortality, liver, and renal effects were observed in F1 animals, although IRIS did not report when these effects were observed with respect to exposure. An extensive number of animal research studies have been conducted on hexachlorobenzene including other developmental studies, but they were not summarized in IRIS.
Other Systemic Toxicity	Splenic, renal, and liver effects were observed in a subchronic dietary exposure study in rats.

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Based on observations of tumors in the liver, thyroid and kidney in three rodent species after oral exposure.
Slope Factor	1.6 per (mg/kg)/day (IRIS; last revised 11/1/96); drinking water unit risk 0.000046 per (ug/L)
Human Data	Inadequate. The reported epidemiological studies of hexachlorobenzene have not been designed to measure increases in cancer incidence as an endpoint and are inadequate in this context.
Animal Data	Hemangiohepatomas, hepatocellular carcinomas and bile duct tumors were significantly increased in treated female rats in a 2-year dietary oral carcinogenicity study; treated males and females had increased incidences of renal cell adenomas and hemangiohepatomas. In a life-time exposure assay in golden hamsters, a significant dose-related increase in the incidence of hepatomas and liver hemangioendotheliomas was observed in males and in females. Treatment-related occurrences of liver tumors were also seen in other oral cancer assays in mice and rats.

alpha-Hexachlorocyclohexane

CHEMICAL STRUCTURE:



CAS NUMBER: 319-84-6

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

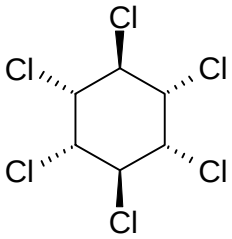
alpha-Hexachlorocyclohexane (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen. Based on increased incidence of liver tumors in five mouse strains and in Wistar rats after dietary exposure to alpha-HCH.
Slope Factor	6.3 per (mg/kg)/day (IRIS; last revised 7/1/93); drinking water unit risk 0.00018 per (ug/L)
Human Data	Available data were inadequate to quantify oral carcinogenicity dose-response.
Animal Data	Dietary alpha-HCH has been shown to cause increased incidences of liver tumors in five mouse strains and in Wistar rats.

gamma-Hexachlorocyclohexane (Lindane)

CHEMICAL STRUCTURE:



CAS NUMBER: 58-89-9

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	0.0003 mg/kg/day (IRIS; last revised 3/1/88)
Critical Effect	Rats receiving 20 and 100 ppm lindane were observed to have greater-than-control incidence of the following: liver hypertrophy, kidney tubular degeneration, hyaline droplets, tubular distension, interstitial nephritis, and basophilic tubules. These effects were mild or rare in animals receiving 4 ppm.
NOAEL	0.33 mg/kg/day (females) (4 ppm in diet). An uncertainty factor of 1000 was used: a factor of 10 each was employed for use of a subchronic vs. a lifetime assay, to account for interspecies variation, and to protect sensitive human subpopulations.
LOAEL	1.55 mg/kg/day (males) (20 ppm in diet)
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	Data on reproductive effects of lindane are inconclusive.
Developmental Toxicity	No data.
Other Systemic Toxicity	Liver effects were also observed in a second oral study in rats. No treatment-related effects were noted on mortality, hematology, clinical chemistry, or urinalysis in the principle study.

gamma-Hexachlorocyclohexane *(Lindane)* ***(continued)***

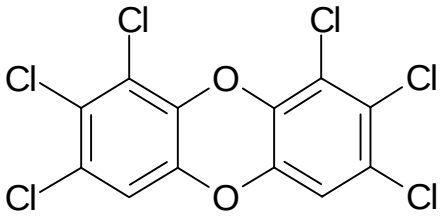
CARCINOGENICITY (U.S. EPA, 1997a; no data available on IRIS)

Weight-of-evidence	B2 or C (unspecified in HEAST).
Slope Factor	1.3 per mg/kg/day (HEAST); oral unit risk 0.000037 ug/L.
Human Data	No data.
Animal Data	Liver tumors were observed in mice in a 2-year dietary exposure assay. No other information was reported in HEAST or IRIS.

1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin

(Synonym of Hexachlorodibenzo-p-dioxin, mixture (HxCDD))

CHEMICAL STRUCTURE:



CAS NUMBER: 19408-74-3

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (continued)

CARCINOGENICITY (U.S. EPA, 2000)

Weight-of-evidence	B2; probable human carcinogen. Based on hepatic tumors in mice and rats exposed by gavage.
Slope Factor	6200 per (mg/kg)/day (IRIS; last revised 3/1/91); drinking water unit risk 0.18 per (ug/L).
Human Data	None. There are no published epidemiologic evaluations of hexachloro- dibenzo-p-dioxin.
Animal Data	The oral slope factor was derived based on the dose-response data of liver tumors (neoplastic nodules, adenomas, and carcinomas) in 2-year gavage studies in mice and rats. No carcinogenic response related to treatment was observed in a mouse skin-painting study.

Lead

CHEMICAL STRUCTURE: Pb

CAS NUMBER: 7439-92-1

TOXICOLOGICAL BASIS for ARAR (U.S. EPA, 1997b): FDA Guidance Value

MEDIA in which ARAR was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 2000a)

RfD	By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/1985 and 07/22/1985) and considered it inappropriate to develop an RfD for inorganic lead. For additional information, interested parties are referred to the 1986 Air Quality Criteria for Lead (EPA-600/8-83/028a-dF) and its 1990 Supplement (EPA/600/8-89/049F). (Last update: 02/01/1991). More current information related to performing lead risk assessments is available at the web page developed by the EPA Technical Review Workgroup for Lead: http://www.epa.gov/superfund/programs/lead/index.htm.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

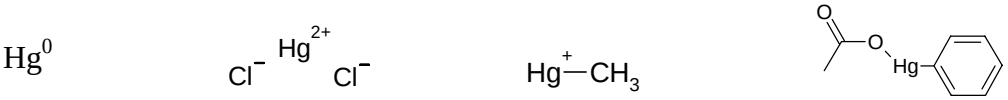
Lead (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	Classification -- B2; probable human carcinogen. Based on sufficient animal evidence. (Last update: 11/01/1993).
Slope Factor	Not available. Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used.
Human Data	Human evidence is inadequate. All of the available epidemiological studies lacked quantitative exposure information, as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium, and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure.
Animal Data	Sufficient. The carcinogenic potential of lead salts (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given lead acetate or subacetate orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. adminstration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetralkyls have not been tested adequately. Studies of inhalation exposure have not been located in the literature.Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression.

Mercury

CHEMICAL STRUCTUREs:



elemental mercury mercuric chloride methyl mercury phenylmercuric acetate

CAS NUMBERs: 7439-97-6 (elemental)
7487-94-7 (mercuric chloride)
22967-92-6 (methyl mercury)
62-38-4 (phenylmercuric acetate)

TOXICOLOGICAL BASIS for ARAR (U.S. EPA, 1997b): Non-cancer effects.

MEDIA in which ARAR was EXCEEDED: Fish

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	Elemental mercury: None. Mercuric chloride: 0.0003 mg/kg-day (This RfD is based on the back calculations from a Drinking Water Equivalent Level (DWEL), recommended to and subsequently adopted by the Agency, of 0.010 mg/L: (RfD = 0.010 mg/L x 2 L/day/70 kg bw = 0.0003 mg/kg bw/day). The LOAEL exposure levels were obtained in the three studies selected as the basis of the recommended DWEL). Methyl mercury: An RfD of 0.0001mg/kg-day based on a NOAEL of for developmental neurological abnormalities in human infants is reported on IRIS for methylmercury (last updated on 05/01/1995). Phenylmercuric acetate: An RfD of 0.00008 based on a NOAEL of 0.0084 mg/kg/day for renal damage in a chronic rat study (last updated 11/01/1996).

Critical Effect	Mercuric chloride: autoimmune effects in Brown Norway rats after subchronic feeding and subcutaneous exposures. Methyl mercury: developmental neurological abnormalities in human infants. Phenylmercuric acetate: renal damage.

Mercury
(continued)

NOAEL	Mercuric chloride: None. Methyl mercury: None. Phenylmercuric acetate: 0.0084 mg/kg-day estimated using the following conversion: food consumption 5% bw/day, molecular weight phenyl mercuric acetate/mercury is 337/201; thus, 0.1 mg/kg of diet (ppm) x 0.05 kg of diet/kg bw/day x 337/201 = 0.0084 mg/kg bw/day. (An ADI of 0.08 ug/kg/day or 6 ug/kg/day for a 70-kg person was derived by dividing the NOEL by an uncertainty factor of 100 to account for species extrapolation and differences in human sensitivity).
LOAEL	Mercuric chloride: 0.226, 0.317, and 0.633 mg/kg-day (an uncertainty factor of 1000 was applied to the animal studies using Brown Norway rats as recommended in U.S. EPA (1987). An uncertainty factor was applied for LOAEL to NOAEL conversion: 10 for use of subchronic studies and a combined 10 for both animal to human and sensitive human populations.) Methyl mercury: Benchmark maternal dose of 1.1 ug/kg-day based estimated from measured maternal body burden (An uncertainty factor of 3 is applied for variability in the human population, in particular the variation in the biological half-life of MeHg and the variation that occurs in the hair:blood ratio for Hg. In addition, a factor of 3 is applied for lack of a two-generation reproductive study and lack of data for the effect of exposure duration on sequelae of the developmental neurotoxicity effects and on adult paresthesia. The total UF is 10.). Phenylmercuric acetate: 0.5 ppm mercury in the diet, or 0.042 mg/kg/day phenyl mercuric acetate for renal damage.

Mercury
(continued)

Human Data

Mercuric chloride: Renal biopsies were performed in 2 (out of 4) workers with nephrotic syndrome who had been occupationally exposed to mercuric oxide, mercuric acetate and probably mercury vapors. Investigators reported that the nephrotic syndrome observed in 3 of the 4 workers may have been an idiosyncratic reaction since many other workers in a factory survey had similarly high levels of urine mercury without developing proteinuria.

Methyl mercury: In 1971-1972 many citizens in rural Iraq were exposed to MeHg-treated seed grain that was mistakenly used in home-baked bread. Latent toxicity was observed in many adults and children who had consumed bread over a 2- to 3-month period. Infants born to mothers who ate contaminated bread during gestation were the most sensitive group. Often infants exhibited neurologic abnormalities while their mothers showed no signs of toxicity. Among the signs noted in the infants exposed during fetal development were cerebral palsy, altered muscle tone and deep tendon reflexes as well as delayed developmental milestones, i.e., walking by 18 months and talking by 24 months. The neurologic signs noted in adults included paresthesia, ataxia, reduced visual fields and hearing impairment. In a report of neurologic abnormalities in four communities of Cree Indians in northern Quebec, a group of 247 children first exhibited clinical signs consistent with MeHg exposure between 12 and 30 months of age. The average indices of exposure were the same for boys and girls at 6 ug/g; only 6% had exposure above 20 ug/g. The prevalence of multiple abnormal neurologic findings was about 4% for children of both sexes. The most frequently observed abnormality was delayed deep tendon reflexes; this was seen in 11.4% of the boys and 12.2% of the girls. These investigators found that when there was a positive association between maternal Hg exposure and abnormal neurologic signs in boys, the incidence rate was 7.2%. Persistence of the Babinski reflex and incoordination due to delayed motor development were seen with equal frequency for both sexes. Other supporting human data are reported in IRIS.

Phenylmercuric acetate: No data.

Immunotoxicity	Mercuric chloride: The most sensitive adverse effect for mercury risk assessment is formation of mercuric-mercury-induced autoimmune glomerulonephritis. The production and deposition of IgG antibodies to the glomerular basement membrane can be considered the first step in the formation of this mercuric-mercury-induced autoimmune glomerulonephritis. The Brown Norway rat should be used for mercury risk assessment. The Brown Norway rat is a good test species for the study of Hg2+-induced autoimmune glomerulonephritis. The Brown Norway rat is not unique in this regard (this effect has also been observed in rabbits). Methyl mercury: No data. Phenylmercuric acetate: No data.

Mercury (continued)

Neurotoxicity	Mercuric chloride: No data. Methyl mercury: An epidemiologic report of MeHg poisoning involved 628 human cases that occurred in Minamata Japan between 1953 and 1960. The overall prevalence rate for the Minamata region for neurologic and mental disorders was 59%. Among this group 78 deaths occurred and hair concentrations of Hg ranged from 50-700 ug/g. The most common clinical signs observed in adults were paresthesia, ataxia, sensory disturbances, tremors, impairment of hearing and difficulty in walking. This particular group of neurologic signs has become known as "Minimata disease." Examination of the brains of severely affected patients that died revealed marked atrophy of the brain (55% normal volume and weight) with cystic cavities and spongy foci. Microscopically, entire regions were devoid of neurons, granular cells in the cerebellum, golgi cells and Purkinje cells. A large database of supporting animal data is reported in IRIS. Phenylmercuric acetate: No data.
Reproductive Toxicity	Mercuric chloride: In male mice administered a single i.p. dose of 1 mg/kg HgCl2, fertility decreased between days 28 and 49 post treatment with no obvious histological effects noted in the sperm. Methyl mercury: No clear reproductive performance effects were reported in IRIS in animal studies. Phenylmercuric acetate: No data.
Developmental Toxicity	Mercuric chloride: No data. Methyl mercury: The initial epidemiologic report of MeHg poisoning involved 628 human cases that occurred in Minamata Japan between 1953 and 1960. Extensive investigations of congenital Minamata disease were undertaken and 20 cases that occurred over a 4-year period were documented. In all instances the congenital cases showed a higher incidence of symptoms than did their mothers. Severe disturbances of nervous function were described and the affected offspring were very late in reaching developmental milestones. Developmental studies in animals also indicated neurological involvement. Phenylmercuric acetate: No data.

Other Systemic Toxicity	Mercuric chloride: In animal studies, nephropathy, proteinurea, altered kidney weights have been reported. Hyperparathyroidism, mineralization of various tissues and fibrous osteodystrophy were observed and considered secondary to the renal impairment. Methyl mercury: chronic nephropathy in animal studies Phenylmercuric acetate: No data.

CARCINOGENICITY (U.S. EPA, 2000a)

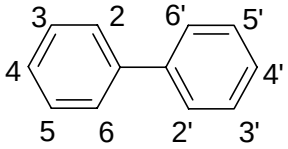
Weight-of-evidence	Elemental mercury: Classification -- D; not classifiable as to human carcinogenicity based on inadequate human and animal data (last updated 05/01/1995). Epidemiologic studies failed to show a correlation between exposure to elemental mercury vapor and carcinogenicity; the findings in these studies were confounded by possible or known concurrent exposures to other chemicals, including human carcinogens, as well as lifestyle factors (e.g., smoking). Findings from genotoxicity tests are severely limited and provide equivocal evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells. Mercuric chloride: Classification -- C; possible human carcinogen based on the absence of data in humans and limited evidence of carcinogenicity in rats and mice (last updated on 06/01/1995). Methyl mercury: Classification -- C; possible human carcinogen based on inadequate data in humans and limited evidence of carcinogenicity in animals (last updated on 05/01/1995). Phenylmercuric chloride: No data.
Slope Factor	Elemental mercury: None. Mercuric chloride: None. Methyl mercury: None.
Human Data	Elemental mercury: Inadequate. A number of epidemiological studies were conducted that examined mortality among elemental mercury vapor-exposed workers. Conflicting data regarding a correlation between mercury exposure and an increased incidence of cancer mortalities have been obtained. All of the studies have limitations that complicate interpretation of their results for associations between mercury exposure and induction of cancer; increased cancer rates were attributable to other concurrent exposures or lifestyle factors. Mercuric chloride: No data. Methyl mercury: Inadequate. Three studies were identified that examined the relationship between methylmercury exposure and cancer. No persuasive evidence of increased carcinogenicity attributable to methylmercury exposure was observed in any of the studies. Interpretation of these studies, however, was limited by poor study design and incomplete descriptions of methodology and/or results.

Mercury
(continued)

Animal Data	Elemental mercury: Inadequate. One study was available that evaluated carcinogenicity of elemental mercury in rats after intraperitoneal injection. Mercuric chloride: Focal papillary hyperplasia and squamous cell papillomas in the forestomach as well as thyroid follicular cell adenomas and carcinomas were observed in male rats gavaged with mercuric chloride for 2 years. The relevance of the forestomach papillomas to assessment of cancer in humans is questionable because no evidence indicated that the papillomas progressed to malignancy. The relevance of the increase in thyroid tumors has also been questioned because these tumors are generally considered to be secondary to hyperplasia; this effect was not observed in the high-dose males. It should also be noted that the authors considered the doses used in the study to exceed the MTD for male rats. In the same study, evidence for increases in squamous cell papillomas in the forestomach of female rats was equivocal. In a second study, equivocal evidence for renal adenomas and adenocarcinomas was observed in male mice; there was a significant positive trend. This tumor type is rare in mice, and the increase in incidence was statistically significant when compared with historic controls. Two other nonpositive lifetime rodent studies were considered inadequate. Mercuric chloride showed mixed results in a number of genotoxicity assays. Methyl mercury: Limited. Three dietary studies in two strains of mice indicate that methylmercury is carcinogenic. Interpretation of two of the positive studies was complicated by observation of tumors only at doses that exceeded the MTD. A fourth dietary study in mice and four dietary studies in rats failed to indicate carcinogenicity associated with methylmercury exposure. Interpretation of four of the nonpositive studies was limited because of deficiencies in study design or failure to achieve an MTD.

PCBs (Total)

CHEMICAL STRUCTURE:



PCBs are a group of compounds which contain chlorine atoms attached to different positions on a biphenyl molecule. Although 209 different congeners can potentially exist, recent studies indicate that the commercial PCBs contained 132 different compounds.

CAS NUMBER: 1336-36-3

TOXICOLOGICAL BASIS for RBC (U.S. EPA, 1999): Cancer effects.

MEDIA in which RBC was EXCEEDED: Fish, Sediment
MEDIA in which ARAR (GDC, 1994) was EXCEEDED: Surface Water

ORAL TOXICITY (U.S. EPA, 1997a, 2000a)

RfD	None.
Critical Effect	No data.
NOAEL	No data.
LOAEL	No data.
Human Data	No data.
Immunotoxicity	No data.
Neurotoxicity	No data.
Reproductive Toxicity	No data.
Developmental Toxicity	No data.
Other Systemic Toxicity	No data.

PCBs (Total) (continued)

CARCINOGENICITY (U.S. EPA, 2000a)

Weight-of-evidence	B2; probable human carcinogen (last revised 6/1/97). Basis -- A 1996 study found liver tumors in female rats exposed to Aroclors 1260, 1254, 1242, and 1016, and in male rats exposed to 1260. These mixtures contain overlapping groups of congeners that, together, span the range of congeners most often found in environmental mixtures. Earlier studies found high, statistically significant incidences of liver tumors in rats ingesting Aroclor 1260 or Clophen A 60. Mechanistic studies are beginning to identify several congeners that have dioxin-like activity and may promote tumors by different modes of action. PCBs are absorbed through ingestion, inhalation, and dermal exposure, after which they are transported similarly through the circulation. This provides a reasonable basis for expecting similar internal effects from different routes of environmental exposure. Information on relative absorption rates suggests that differences in toxicity across exposure routes are small. The human studies are being updated; currently available evidence is inadequate, but suggestive.
Slope Factor	For high risk sub-populations, the following slope factors for polychlorinated biphenyls were provided in IRIS: upper-bound slope factor - 2.0 per (mg/kg)/day; central-estimate slope factor - 1.0 per (mg/kg)/day. Highly exposed populations include some nursing infants and consumers of game fish, game animals, or products of animals contaminated through the food chain. The criteria for using slope factors for high risk populations include food chain exposure and sediment or soil ingestion. The slope factors were based on reported incidences of liver hepatocellular adenomas, carcinomas, cholangiomas, or cholangiocarcinomas in female Sprague-Dawley rats after dietary exposures.
Human Data	Inadequate for quantifying risk of cancer in humans after PCB exposure.
Animal Data	Increased incidences of liver adenomas and carcinomas and/or thyroid adenomas or carcinomas in rats after chronic dietary exposure to Aroclor 1260 and Aroclor 1254.

Appendix B REFERENCES

Bodek, I., W.J. Lyman, W.F. Reehl, and D.H. Rosenblatt. 1988. Environmental Inorganic Chemistry: Properties, Processes, and Estimation Methods. SETAC Special Publication Series. Pergamon Press, New York.

Government of the District of Columbia (GDC). 1994. Water Quality Standards, Department of Consumer and Regulatory Affairs. March 4. 41D.C. Reg.1075.

U.S. EPA. 1989. Interim Procedures of Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CDD and CDFs) and 1989 Update. Risk Assessment Forum, U.S. Environmental Protection Agency. EPA/625/3-89/016, PB90-145756.

U.S. EPA. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Final Draft. ECAO-CIN-842. March, 1993.

U.S. EPA. 1997a. Health Effects Assessment Summary Tables (HEAST). FY 1997 Update. EPA-540-R-997-036.

U.S. EPA. 1997b. The Incidence and Severity of Sediment Contamination in Surface Waters of the United States. Vol 1: National Sediment Quality Survey. Office of Science and Technology. EPA 823-R-97-006.

U.S. EPA. 1998. National Recommended Water Quality Criteria. December 10, 1998. 63(237)FR 68354-68364.

U.S. EPA. 1999. Risk-Based Concentration Tables. Memorandum from Jennifer Hubbard, Region III U.S. EPA to RBC Table Users.

U.S. EPA. 2000a. Integrated Risk Information System (IRIS). Online. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 2000b. The US EPA TEF Values. Office of Research and Development, U.S. Environmental Protection Agency web page (http://www/epa/gov/nceawww1/dchem.htm).

APPENDIX C - RAGS D TABLES

TABLE C-2.2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
TIDAL ANACOSTIA RIVER

Scenario Timeframe:	**Current**
Medium:	**Various**
Exposure Medium:	**Fish Tissue**
Exposure Point:	**N/A**

CAS Number	Chemical	Minimum Concentration[1]	Minimum Qualifier[2]	Maximum Concentration[1]	Maximum Qualifier[2]	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits[3]	Concentration Used for Screening[4]	Background Value[5]	Screening Toxicity Value[6]	Potential ARAR/ TBC Value	Potential ARAR/ TBC Source[7]	COPC Flag	Rationale for Contaminant Deletion or Selection[8]
83329	Acenaphthene	6.30E-04	J	2.83E-02		PPM	LA	12 / 29	0.0003 - 0.002	2.83E-02	N/A	8.11E+00 N	6.50E+01	2	YES	IEI
208968	Acenaphthylene	3.50E-04	J	5.00E-03		PPM	LA	12 / 29	0.0004 - 0.0009	5.00E-03	N/A				YES	IEI
309002	Aldrin	2.90E-04		2.31E-03		PPM	LA	8 / 32	0.0001 - 0.001	2.31E-03	N/A	1.86E-04 C	6.30E-04	1	YES	ATL
120127	Anthracene	8.00E-04		1.23E-02		PPM	LA	12 / 29	0.0003 - 0.004	1.23E-02	N/A	4.06E+01 N	3.20E+02	2	YES	IEI
11096825	Aroclor 1260	1.80E-01		4.50E-01		PPM	16330	3 / 3		4.50E-01	N/A	1.58E-03 C	1.40E-03	1	YES	ATL
7440382	Arsenic	2.50E-02		2.66E-01		PPM	LA	10 / 16	0.029 - 0.05	2.66E-01	N/A	2.10E-03 C	6.20E-03	1	YES	ATL
117817	Bis(2ethylhexyl) phthalate	5.80E-02		6.40E-01		PPM	3	16 / 18	0.009 - 0.009	6.40E-01	N/A	2.25E-01 C	7.70E-01	1	YES	ATL
56553	Benz(a)anthracene	2.00E-04	J	1.35E-03		PPM	LA	11 / 29	0.0004 - 0.005	1.35E-03	N/A	4.32E-03 C	1.50E-02	1	YES	IEI
53703	Dibenz(a,h)anthracene	1.00E-05	J	1.20E-04	J	PPM	LA	11 / 29	0.001 - 0.002	1.20E-04	N/A	4.32E-04 C	1.50E-03	1	YES	IEI
50328	Benzo(a)pyrene	7.00E-05	J	2.60E-04	J	PPM	KM	11 / 29	0.0006 - 0.002	2.60E-04	N/A	4.32E-04 C	1.50E-03	1	YES	IEI
205992	Benzo(b)fluoranthene	4.00E-05	J	4.90E-04	J	PPM	KM	11 / 29	0.0005 - 0.003	4.90E-04	N/A	4.32E-03 C	1.50E-02	1	YES	IEI
192972	Benzo(e)pyrene	1.10E-04	J	6.40E-04		PPM	UA	11 / 11		6.40E-04	N/A				YES	IEI
191242	Benzo(g,h,i)perylene	7.00E-05	J	3.10E-04	J	PPM	LA	11 / 29	0.0005 - 0.005	3.10E-04	N/A				YES	IEI
92524	Biphenyl	6.90E-04	J	1.06E-01		PPM	LA	11 / 11		1.06E-01	N/A	6.76E+00 N	5.40E+01	2	YES	IEI
207089	Benzo(k)fluoranthene	6.00E-05	J	1.60E-04	J	PPM	LA	11 / 29	0.0005 - 0.003	1.60E-04	N/A	4.32E-02 C	1.50E-01	1	YES	IEI
7440439	Cadmium	3.02E-03		2.00E-01		PPM	16330	15 / 16	0.003 - 0.003	2.00E-01	N/A	1.35E-01 N	5.40E-01	2	YES	ATL
5103742	Transchlordane	3.90E-02		2.30E-01		PPM	LA	10 / 12		2.30E-01	N/A		8.30E-03	1	YES	ASL
5103719	Cischlordane	2.00E-03		3.40E-01		PPM	4	38 / 41		2.30E-01	N/A		8.30E-03	1	YES	ASL
27304138	Oxychlordane	1.09E-03		1.00E-02		PPM	BRA	12 / 23		1.00E-02	N/A				YES	IEI
5566347	Chlordane gamma	5.00E-04		9.00E-02		PPM	3	29 / 29		9.00E-02	N/A		8.30E-03	1	YES	ASL
7440473	Chromium, total	4.71E-02		5.00E-01		PPM	16330	10 / 16	0.04 - 0.5	5.00E-01	N/A		5.40E+00	2	YES	IEI
218019	Chrysene	2.60E-04	J	5.37E-03		PPM	LA	11 / 29	0.0004 - 0.005	5.37E-03	N/A	4.32E-01 C	1.50E+00	1	YES	IEI
319846	Hexachlorocyclohexanealpha	1.10E-04		8.00E-03		PPM	16330	13 / 32	0.00009 - 0.002	8.00E-03	N/A	5.01E-04 C	1.70E-03	1	YES	ATL
319857	Hexachlorocyclohexanebeta	2.00E-05	J	5.00E-04		PPM	LA	6 / 29	0.00003 - 0.003	5.00E-04	N/A	1.75E-03 C	6.00E-03	1	YES	IEI
319868	Hexachlorocyclohexanedelta	4.90E-04		4.90E-04		PPM	LA	1 / 29	0.00016 - 0.002	4.90E-04	N/A		6.00E-03	1	YES	IEI
58899	Hexachlorocyclohexanegamma (Lindane)	1.60E-04		2.58E-03		PPM	LA	12 / 32	0.0003 - 0.002	2.58E-03	N/A	2.43E-03 C	8.30E-03	1	YES	ATL
118741	Hexachlorobenzene (HCB)	2.50E-04		4.98E-03		PPM	LA	13 / 32	0.0005 - 0.004	4.98E-03	N/A	1.97E-03 C	6.70E-03	1	YES	ATL
7440508	Copper	2.70E-01		7.50E-01		PPM	16330	5 / 5		7.50E-01	N/A	5.41E+00 N	4.00E+01	2	YES	IEI
1861321	Dacthal	1.00E-03		1.00E-03		PPM	16330	1 / 3	0.001 - 0.001	1.00E-03	N/A	1.35E+00 N	1.10E+01	2	YES	IEI
84662	Diethyl phthalate	4.00E-03		1.40E-02		PPM	4	8 / 18	0.001 - 0.002	1.40E-02	N/A	1.08E+02 N	8.60E+02	2	YES	IEI
132650	Dibenzothiophene	3.10E-04	J	1.53E-02		PPM	LA	11 / 11		1.53E-02	N/A				YES	IEI
60571	Dieldrin	2.50E-04		5.20E-02		PPM	LA	41 / 44		5.20E-02	N/A	1.97E-04 C	6.70E-04	1	YES	ATL
84742	Dinbutyl phthalate	2.50E-02		1.60E-01		PPM	3	17 / 18	0.005 - 0.005	1.60E-01	N/A	1.35E+01 N	1.10E+02	2	YES	IEI
33213659	Endosulfanbeta	3.40E-04		4.10E-04		PPM	KM	2 / 29	0.00015 - 0.001	4.10E-04	N/A		6.50E+00	2	YES	IEI
1031078	Endosulfan sulfate	4.00E-03		4.00E-03		PPM	3	1 / 18	0.0008 - 0.001	4.00E-03	N/A				YES	IEI
72208	Endrin	9.90E-04		1.12E-03		PPM	LA	2 / 32	0.00018 - 0.002	1.12E-03	N/A	4.06E-02 N	3.20E-01	2	YES	IEI
206440	Fluoranthene	5.00E-04	J	3.06E-02		PPM	UA	14 / 29	0.0004 - 0.002	3.06E-02	N/A	5.41E+00 N	4.30E+01	2	YES	IEI
86737	Fluorene	8.90E-04	J	6.01E-02		PPM	LA	13 / 29	0.0003 - 0.002	6.01E-02	N/A	5.41E+00 N	4.30E+01	2	YES	IEI
1024573	Heptachlor epoxide	5.90E-04		1.70E-02		PPM	4	26 / 32	0.0005 - 0.001	1.70E-02	N/A	3.47E-04 C	1.20E-03	1	YES	ATL

TABLE C-2.2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
TIDAL ANACOSTIA RIVER

Scenario Timeframe:	**Current**
Medium:	**Various**
Exposure Medium:	**Fish Tissue**
Exposure Point:	**N/A**

CAS Number	Chemical	Minimum Concentration[1]	Minimum Qualifier[2]	Maximum Concentration[1]	Maximum Qualifier[2]	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits[3]	Concentration Used for Screening[4]	Background Value[5]	Screening Toxicity Value[6]	Potential ARAR/ TBC Value	Potential ARAR/ TBC Source[7]	COPC Flag	Rationale for Contaminant Deletion or Selection[8]
76448	Heptachlor (pesticide)	5.00E-05	J	6.10E-03		PPM	3	12 / 32	0.00012 - 0.001	6.10E-03	N/A	7.01E-04 C	2.40E-03	1	YES	ATL
193395	Indeno(1,2,3c,d)pyrene	4.00E-05	J	2.00E-04	J	PPM	LA	11 / 29	0.005 - 2	2.00E-04	N/A	4.32E-03 C	1.50E-02	1	YES	IEI
78591	Isophorone	1.90E+00		1.90E+00		PPM	4	1 / 18	0.0004 - 0.005	1.90E+00	N/A	3.32E+00 C	1.10E+01	1	YES	IEI
7439921	Lead	2.50E-02		4.20E+00		PPM	16330	16 / 16		4.20E+00	N/A		1.30E+00	3	YES	ASL
7439965	Manganese	5.50E-01		6.00E-01		PPM	ANA82	2 / 2		6.00E-01	N/A	1.89E+01 N	5.40E+00	2	YES	IEI
2245387	1,6,7Trimethylnaphthalene	8.20E-04	J	2.09E-01		PPM	LA	11 / 11		2.09E-01	N/A				YES	IEI
7439976	Mercury	2.49E-02		1.59E-01		PPM	16330	16 / 16		1.59E-01	N/A	1.35E-02 N	1.10E-01	2	YES	ATL
90120	1Methylnaphthalene	2.29E-03		1.84E-01		PPM	LA	11 / 11		1.84E-01	N/A				YES	IEI
91576	2Methylnaphthalene	3.64E-03		2.72E-01		PPM	LA	11 / 11		2.72E-01	N/A	2.70E+00 N			YES	IEI
581420	2,6Dimethylnaphthalene	1.10E-03	J	3.82E-01		PPM	LA	11 / 11		3.82E-01	N/A				YES	IEI
832699	1Methylphenanthrene	2.50E-04	J	1.34E-02		PPM	LA	11 / 11		1.34E-02	N/A				YES	IEI
2385855	Mirex (pesticide = dechlorane)	8.00E-05		8.80E-04		PPM	UA	11 / 14	0.001 - 0.001	8.80E-04	N/A	2.70E-02 N	6.00E-03	1	YES	IEI
91203	Naphthalene	4.80E-03		1.60E-01		PPM	3	25 / 29	0.0001 - 0.0001	1.60E-01	N/A	2.70E+00 N	4.30E+01	2	YES	IEI
7440020	Nickel	3.40E-02		7.16E-02		PPM	LA	6 / 13	0.03 - 0.05	7.16E-02	N/A	2.70E+00 N	2.20E+01	2	YES	IEI
117840	DiNoctyl phthalate	3.90E-02		6.70E+00		PPM	4	16 / 18	0.007 - 0.03	6.70E+00	N/A	2.70E+00 N	2.20E+01	2	YES	ATL
39765805	Trans nonachlor	1.07E-02		3.70E-01		PPM	LA	21 / 23		3.70E-01	N/A				YES	IEI
5103731	cisNonachlor	4.44E-03		8.20E-02		PPM	LA	13 / 23		8.20E-02	N/A				YES	IEI
3268879	Octachlorodibenzopdioxin	6.50E-07		5.71E-05		PPM	4	18 / 18		5.71E-05	N/A	2.10E-05 C			YES	ATL
39001020	Octachlorodibenzofuran	1.00E-07		9.22E-05		PPM	3	18 / 18		9.22E-05	N/A	2.10E-05 C			YES	ATL
53190	o,p'DDD	2.37E-03		1.43E-02		PPM	UA	11 / 11		1.43E-02	N/A		4.50E-02	1	YES	IEI
3424826	o,p'DDE	9.00E-05	J	1.27E-03		PPM	LA	11 / 11		1.27E-03	N/A		3.20E-02	1	YES	IEI
789026	o,p'DDT	6.20E-04		6.90E-03		PPM	LA	10 / 11	0.00008 - 0.00008	6.90E-03	N/A		3.20E-02	1	YES	IEI
1336363	PCBS, total	4.07E-02		4.60E+00		PPM	LA	43 / 44		4.60E+00	N/A	1.578E-02 C	1.40E-03	1	YES	ATL
35822469	H7CDD1234678	1.00E-07		6.20E-06		PPM	4	15 / 18	0.0000002 - 0.0000073	6.20E-06	N/A	2.10E-06 C			YES	ATL
39227286	H6CDD123478	1.00E-07		5.70E-06		PPM	3	16 / 18	0.0000001 - 0.00000085	5.70E-06	N/A	2.10E-07 C			YES	ATL
57653857	H6CDD123678	5.00E-08		7.40E-06		PPM	3	16 / 18	0.00000015 - 0.00000075	7.40E-06	N/A	2.10E-07 C			YES	ATL
40321764	PCDD12378	5.00E-08		3.90E-06		PPM	4	16 / 18	0.0000002 - 0.0000016	3.90E-06	N/A	4.20E-07 C			YES	ATL
19408743	H6CDD123789	5.00E-08		1.03E-05		PPM	3	17 / 18	0.00000085 - 0.00000085	1.03E-05	N/A	2.09E-07 C			YES	ATL
1746016	TCDD2378 (dioxin)	5.00E-08		2.80E-06		PPM	3	18 / 18		2.80E-06	N/A	2.10E-08 C	6.90E-08	1	YES	ATL
67562394	H7CDF1234678	5.00E-08		1.96E-05		PPM	3	18 / 18		1.96E-05	N/A	2.10E-06 C			YES	ATL
70648269	H6CDF123478	5.00E-08		1.00E-05		PPM	3	18 / 18		1.00E-05	N/A	2.10E-07 C			YES	ATL
55673897	H7CDF1234789	5.00E-08		2.55E-06		PPM	4	18 / 18		2.55E-06	N/A	2.10E-06 C			YES	ATL
57117449	H6CDF123678	5.00E-08		8.10E-06		PPM	3	18 / 18		8.10E-06	N/A	2.10E-07 C			YES	ATL
57117416	PCDF12378	5.00E-08		5.00E-06		PPM	3	17 / 18	0.0000007 - 0.0000007	5.00E-06	N/A	4.20E-06 C			YES	ATL
72918219	H6CDF123789	1.00E-07		9.50E-06		PPM	3	16 / 18	0.00000015 - 0.00000025	9.50E-06	N/A	2.10E-07 C			YES	ATL
60851345	H6CDF234678	5.00E-08		5.00E-06		PPM	3	17 / 18	0.0000045 - 0.0000045	5.00E-06	N/A	2.10E-07 C			YES	ATL
57117314	PCDF23478	5.00E-08		4.75E-06		PPM	3	18 / 18		4.75E-06	N/A	4.20E-07 C			YES	ATL
51207319	TCDF2378	5.00E-08		4.80E-06		PPM	3	18 / 18		4.80E-06	N/A	2.10E-07 C			YES	ATL
198550	Perylene	8.00E-05	J	4.90E-04	J	PPM	LA	11 / 11		4.90E-04	N/A				YES	IEI

TABLE C-2.2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
TIDAL ANACOSTIA RIVER

Scenario Timeframe:	**Current**
Medium:	**Various**
Exposure Medium:	**Fish Tissue**
Exposure Point:	**N/A**

CAS Number	Chemical	Minimum Concentration[1]	Minimum Qualifier[2]	Maximum Concentration[1]	Maximum Qualifier[2]	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits[3]	Concentration Used for Screening[4]	Background Value[5]	Screening Toxicity Value[6]	Potential ARAR/ TBC Value	Potential ARAR/ TBC Source[7]	COPC Flag	Rationale for Contaminant Deletion or Selection[8]
85018	Phenanthrene	1.35E-03		1.03E-01		PPM	LA	15 / 29	0.0003 - 0.004	1.03E-01	N/A				YES	IEI
108952	Phenol	4.00E-02		4.00E-02		PPM	4	1 / 18	0.001 - 0.01	4.00E-02	N/A	8.11E+01 N	6.50E+02	2	YES	IEI
72548	p,p'DDD	1.00E-03		4.80E-01		PPM	LA	42 / 44		4.80E-01	N/A	1.31E-02 C	4.50E-02	1	YES	ATL
72559	p,p'DDE	3.70E-03		5.00E-01		PPM	LA	43 / 44		5.00E-01	N/A	9.28E-03 C	3.20E-02	1	YES	ATL
50293	p,p'DDT	5.00E-04		5.10E-02		PPM	LA	30 / 44	0.001 - 0.001	5.10E-02	N/A	9.28E-03 C	3.20E-02	1	YES	ATL
129000	Pyrene	4.90E-04	J	3.30E-02		PPM	4	14 / 29	0.0003 - 0.003	3.30E-02	N/A	4.06E+00 N	3.20E+01	2	YES	IEI
7782492	Selenium	8.14E-02		5.04E-01		PPM	LA	11 / 11		5.04E-01	N/A	6.76E-01 N	5.40E+00	2	YES	IEI
7440224	Silver	2.50E-02		2.50E-02		PPM	ANA82	2 / 13	0.004 - 0.007	2.50E-02	N/A	6.76E-01 N	5.40E+00	2	YES	IEI
57749	Total chlordane (alpha+cis+oxy+trans)	8.00E-02		8.00E-01		PPM	LA	10 / 12		8.00E-01	N/A	9.01E-03 C	8.30E-03	1	YES	ATL
7440666	Zinc	7.48E-01		2.37E+01		PPM	16330	16 / 16		2.37E+01	N/A	4.06E+01 N	3.20E+02	2	YES	IEI

(1) Minimum/maximum detected concentration.

(2) J = estimated concentration

(3) Blank indicates detection limits were not available

(4) Maximum concentration used as screening value.

(5) N/A - Information on background concentrations was not available.

(6) U.S. EPA. 1999. Risk-Based Concentration Tables. Memorandum from Jennifer Hubbard, Region III U.S. EPA to RBC Table Users. (Cancer benchmark value = 1E-06, HQ = 0.1)

(7) U.S. EPA. 1997. The Incidence and Severity of Sediment Contamination in Surface Waters of the United States. Vol 1: National Sediment Quality Survey. Office of Science and Technology. EPA 823-R-97-006.

1E-06

2 - Noncancer based; HQ = 0.1

3 - FDA guidance/action/tolerance level

(8) Rationale Codes:
Above Toxicity Level (ATL)
Above Screening Levels (ASL)
Insufficient Exposure Information (IEI)
Insufficient Toxicity Information (ITI)

Definitions: N/A = Not Available
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requiremer
C = Carcinogenic
N = Non-Carcinogenic